619
C81
1925/26
cop. 2

Legislative Document (1927) No. 28

STATE OF NEW YORK

REPORT

OF THE

New York State Veterinary College

At Cornell University

For the Year 1925-1926

THE LIBRARY OF THE
MAY 25 1927
UNIVERSITY OF ILLINOIS

ALBANY
J. B. LYON COMPANY, PRINTERS
1927

Legislative Document (1927) No. 28

STATE OF NEW YORK

REPORT

OF THE

New York State Veterinary College

At Cornell University

For the Year 1925-1926

THE LIBRARY OF THE
MAY 25 1927
UNIVERSITY OF ILLINOIS

ALBANY
J. B. LYON COMPANY, PRINTERS
1927

619
C81
1925/26
cop.2

CORNELL UNIVERSITY

ITHACA, NEW YORK

August 5, 1926.

The Honorable ALFRED E. SMITH, *Governor of the State of New York, Albany, N. Y.:*

SIR.—The act of the Legislature of the State of New York, approved May 22, 1897 (Chap. 689 of the Laws of 1897), providing for the administration of the New York State Veterinary College at Cornell University, requires that the said University shall make an annual report to the Governor of the expenditures and general operations of the college. In accordance with this mandate, I have the honor to submit, on behalf of Cornell University, the accompanying report of the College for the fiscal year 1925–26.

The report gives a detailed account of the work of the college for the past year. It calls attention to the need of larger appropriations for research in animal diseases and points out certain heretofore unknown relations between infections of animals and those of man. The economic and sanitary importance of competent veterinary service to the animal husbandry of the State, justifies the request of the Dean for liberal appropriations for research and the teaching activities of the college.

Respectfully submitted,

LIVINGSTON FARRAND
President.

25 May 27 div.g. 1925/26 cvl. EH

TABLE OF CONTENTS

PAGE

Letter of Submittal 3
Report of the Dean 7
Appendix:
Report of the Ambulatory Clinic 27
Report of the Surgical and Consulting Clinics 31
Report of the Small Animal Clinic 33
Report of the Diagnosis Laboratory 36
Report of the Diagnostic Work on Poultry Diseases:
a. Ithaca 50
b. Farmingdale 54
The Interpretation of Clinical Symptoms. D. H. Udall 61
Convulsions in Dogs. H. J. Milks 70
A Comparison of the Blood of a Normal and Two Castrated Billy Goats. P. A. Fish and C. E. Hayden 76
The Agglutination Test as an Aid in Handling Bang Abortion Disease. R. R. Birch and H. L. Gilman 81
Bang Abortion Disease in Cattle. R. R. Birch and H. L. Gilman 86
Technique of Drawing Blood Samples from Cattle for the Agglutination Test. R. R. Birch 103
Immunizing Young Pigs against Hog Cholera. J. W. Benner 106
The Passage of Bacterium Abortum through the Digestive Tract of Cattle. H. L. Gilman and R. R. Birch 118
Directions for Collecting Blood Samples for the Agglutination Test for Infectious Abortion. C. M. Carpenter 121
The Value of Placental Examination for the Diagnosis of Infectious Abortion of Cattle. W. A. Hagan 125
Bacillary White Diarrhea. E. L. Brunett 131
The Differentiation of *Bact. pullorum* (Rettger) and *Bact. sanguinarium* (Moore). J. M. Hendrickson 146
Undulant Fever in Man Associated with Bacteria Indistinguishable from *Brucella abortus*. Veranus A. Moore and C. M. Carpenter 173
Agglutinins for *Brucella abortus* in the Blood Serum of Man. C. M. Carpenter 178
A Comparison of Strains of *Brucella abortus* Isolated from Man with Those from Cattle. C. M. Carpenter 182
Growth of Veterinary Science and the Application of New Knowledge. Veranus A. Moore 186

REPORT

ITHACA, N. Y., *August* 5, 1926.

President LIVINGSTON FARRAND:

SIR.— I have the honor to submit the appended report of the New York State Veterinary College at Cornell University for the fiscal year ending June 30, 1926.

The year has been an unusually active one for the instructing staff. The veterinary course is becoming more difficult to cover in the allotted time, owing to the rapid development of new knowledge, the appearance of new diseases and the extension of the service. The curriculum requires constant readjustments, and always with care that the newer findings, important as they may be, are not substituted for essential and fundamental principles which they supplement, but do not replace. The researches in animal diseases have been continued to the full extent permitted by the appropriations. Members of the faculty have done all they could by way of consultations and conferences to aid veterinary practitioners in the State.

A deeper interest has been manifested on the part of animal owners in veterinary service than in former years. The reports of the college have been in greater demand by veterinarians, and the requests from educational and research institutions for graduates as assistants, and from communities for practitioners, have been in excess of the number that could be filled from the senior class. There are strong indications that a real need exists for a larger number of veterinarians than are being graduated. The profession is becoming adjusted to the changes that have taken place in the methods for the control of infectious diseases. Animal owners are coming to realize in this country, as they do in Europe, that trained veterinarians are counselors they should consult relative to matters pertaining to the prevention and treatment of diseases.

The Federal Bureau of Animal Industry estimates that the losses in the United States, from communicable diseases alone, approximate $200,000,000 annually.* If we add to that large

*Hog cholera	30	to	50 million	dollars
Tuberculosis	35	to	40 million	dollars
Southern cattle tick	20	to	30 million	dollars
Contagious abortion	20	to	30 million	dollars
Hemorrhagic septicemia	15	to	20 million	dollars
Influenza	15	to	20 million	dollars
Plant poisoning	15	to	20 million	dollars
Blackleg			6 million	dollars
Scabies of sheep and cattle			4 million	dollars
Anthrax			2 million	dollars
Dourine	8	to	10 thousand	dollars
Glanders	8	to	10 thousand	dollars

sum the losses due to sporadic diseases for which the services of veterinarians are in greatest demand, and for which there are no accurate statistics, the loss sustained by animal owners could be more accurately determined. Many practitioners in this State report that the general or sporadic diseases are the cause of from 75 to 95 per cent of the deaths from disease. The distribution of competent veterinarians through the live stock districts is the greatest protection against these losses. Veterinarians have large responsibilities and for success in their efforts they must have the cooperation of animal owners.

The new discoveries in connection with the diagnosis and prevention of Bang abortion disease; the rapidly growing poultry industry and the heavy losses it sustains from disease; the public health need for more extended municipal meat and dairy inspection; the increased amount of tuberculin testing of accredited herds required to assure their continuous freedom from tuberculosis; and the ever-growing demand for veterinary service in connection with small animals, all point to the increasing opportunities in veterinary medicine for rendering service of a distinct and highly valuable nature. Researches are revealing new facts concerning the nature of many disorders among animals and bringing out important methods for their diagnosis and prevention. These findings are adding to the attractiveness of veterinary medicine. The discovery of the close relationship between certain diseases of animals — such as tuberculosis, the Bang abortion disease, tularaemia and swine erysipelas — and those of man emphasizes the fact that veterinarians have not only to deal with maladies involving economic loss, but also that they have important responsibilities to the public.

FACULTY

A few changes have occurred in the instructing staff. Dr. S. A. Goldberg has accepted an appointment as pathologist in the Bronx Hospital in New York City; Dr. E. R. Cushing has resigned to accept an attractive position with the H. K. Mulford Company, Philadelphia; Dr. H. B. McMurray succeeded Dr. M. H. Mabey as instructor in surgery. Dr. W. A. Hagan was on sabbatic leave during the first semester. He spent the time in Europe studying methods of teaching bacteriology and research in animal diseases. At the spring meeting of the trustees he was promoted to the head of the Department of Pathology, Bacteriology and Meat Inspection. Dr. R. R. Birch was on sabbatic leave the latter half of the year. He is in Europe studying the Bang abortion disease and methods for controlling it. He will return early in the fall.

In the summer of 1925, the International Education Board granted fellowships to Drs. Hagan and Birch for studying abroad. It is very gratifying that this Board has recognized the scientific significance of veterinary subjects and is tendering most helpful assistance in advancing knowledge concerning them. Dr. Franz

Benesch, professor of obstetrics in the Veterinary College in Vienna, Austria, is spending several months at this college on a similar fellowship. He has brought us many valuable suggestions and likewise expresses himself appreciative of the aid he has received here, especially from the ambulatory clinic. This splendid opportunity for young teachers in veterinary colleges of America and Europe to study in other lands promises to be of much direct benefit to veterinary science and practice, and indirectly to animal owners.

As heretofore, the University has furnished in its respective departments instruction to veterinary students in chemistry, embryology and histology and the College of Agriculture has instructed them in animal husbandry, botany and zoology. Reciprocally, the veterinary college has given instruction in bacteriology, pathology, physiology and horseshoeing, aggregating 387 university hours, to 187 students registered in other colleges of the university. Further, a special three-hour course on "Health and Diseases of Domesticated Animals" was given in the first term for students in the College of Agriculture, especially those in the Department of Animal Husbandry.

Personnel of the Teaching Staff

Livingston Farrand, president of the University.
Simon Henry Gage, emeritus professor of histology.
Walter Long Williams, emeritus professor of obstetrics and research in the diseases of breeding animals.

Department of Anatomy

Dr. Grant S. Hopkins, professor of anatomy.
Dr. Earl Sunderville, assistant professor and secretary of the faculty.
Mr. André R. DeMott, student assistant.

Department of Materia Medica

Dr. Howard J. Milks, professor of therapeutics and small animal clinic.
Dr. H. C. Stephenson, assistant professor.

Department of Medicine

Dr. Dennie H. Udall, professor of veterinary medicine and hygiene and superintendent of the ambulatory clinic.
Dr. M. G. Fincher, assistant professor.
Dr. E. R. Cushing, instructor.

Department of Pathology and Bacteriology

Dr. V. A. Moore, professor of comparative pathology and bacteriology and dean of the college.
Dr. W. A. Hagan, professor of bacteriology and parasitology.
Dr. S. A. Goldberg, assistant professor of pathology.
Dr. C. M. Carpenter, assistant professor of diagnosis.
Dr. E. L. Brunett, instructor in poultry diseases.
Dr. J. M. Hendrickson, instructor in poultry diseases.
Mr. Peter Olafson, instructor in pathology.
Mr. Charles J. Parshall, assistant in diagnosis.
Dr. H. M. DeVolt, assistant in poultry diseases.
Mr. Alexander Zeissig, student assistant in bacteriology.

Department of Physiology

Dr. Pierre A. Fish, professor of veterinary physiology and curator of museum.
Dr. C. E. Hayden, assistant professor.

Department of Surgery

Dr. J. N. Frost, professor of veterinary surgery.
Dr. H. B. McMurray, instructor.

Veterinary Experiment Station

Dr. Raymond R. Birch, superintendent of the station.
Dr. J. W. Benner, assistant professor.
Dr. H. L. Gilman, assistant professor.

Farriery

Mr. Henry Asmus, assistant professor of horseshoeing.

Library

Miss E. Clifford Williams, librarian of the Roswell P. Flower library.

Business Office

Miss Helena H. Haight, clerk and bookkeeper.
Mrs. James A. Washburn, stenographer and office secretary.
Miss Eileen Carey, clerk and stenographer.

Machinist

Mr. Archibald Wilson.

Fourteen men were employed as grooms, attendants and janitors. In addition, the college had an appropriation of $2,400 for special labor and assistants during crowded parts of certain courses and necessary assistance in laboratory courses, diagnosis work and the preparation of biologic diagnosis and prophylactic material.

NON-RESIDENT LECTURERS

The college was fortunate in securing non-resident lecturers. Dr. W. H. Park, Director of the Research Laboratory of the New York City Board of Health, spoke on "Immunization against Bovine Tuberculosis and its Relation to Human Tuberculosis"; Dr. L. Van Es of the University of Nebraska gave an address on "Avian Tuburculosis and Its Relation to this Disease in other Species of Animals"; Dr. M. Dorset of the Federal Bureau of Animal Industry discussed the subject of "Immunization of Young Pigs against Hog Cholera"; C. H. Case of Akron, Ohio, presented "The Treatment of Diseases of the Genital Organs"; Commissioner B. A. Pyrke of the Department of Farms and Markets, Albany, spoke on "The Cooperation of the Department of Farms and Markets in Veterinary Medicine," and Dr. W. L. Williams, Professor Emeritus, of Ithaca, on "The Genital Diseases of Horses." These were all very instructive and helpful to students

and practitioners. Lectures on practical points in connection with veterinary practice, jurisprudence and professional ethics were given by Drs. F. E. McClelland of Buffalo, President of the State Veterinary Society, R. W. Gannett of Brooklyn, Ira Buchanan of Auburn, W. G. Hollingworth of Utica, E. V. Moore of Cortland and Professors L. A. Maynard and J. D. Brew of the College of Agriculture and Professor L. P. Wilson of the Law School. These men brought much useful information for students who are about to enter active practice.

STUDENTS

In previous reports attention has been called to the reduction in the number of veterinary students in this country since the World War. The apparent reasons for this decline have been enumerated. In many European countries there has been a larger registration in veterinary colleges than formerly. In Denmark, for example, with a cattle population slightly larger than that of New York State, there is an enrollement of 300 veterinery students.

Although the economic conditions that embarrassed practitioners have improved and many herds of cattle are being accedited by the government and turned over to local practitioners for annual tests, the students in the veterinary colleges were slightly fewer than in 1924–1925. It is gratifying to report that, notwithstanding a smaller total, this college had an increase of ten per cent, and in the freshman class twenty per cent over the registration of 1925. There were enrolled a total of 89 undergraduate students, distributed by classes as follows: 24 freshmen, 18 sophomores, 26 juniors and 21 seniors. There were two graduate veterinarians working for an advanced degree and four who registered in the practitioners' course. The college enrolled 17 per cent of the veterinary students in the United States.

There is a shortage in the supply of graduate veterinarians in this country, but it undoubtedly will be temporary. Unfortunately for all concerned, there are men who occupy responsible positions educationally who have conceived the idea that teachers of agriculture in high schools, because they have had training in certain somewhat closely allied sciences, are competent to teach and advise farmers how to treat their sick and injured animals. However, the verdict of disappointment is being rendered by many of those who have applied such teaching. It should be remembered that advice in treating diseases given to those "who cannot make the diagnosis" may be so applied as to kill more than it cures, and, further, that free service may spread the spirit of pauperism among the well-to-do as well as give aid to the needy. Why should men spend years in studying veterinary medicine if those who have had courses in other subjects can treat diseases and wisely advise animal owners relative to them? There seems to be no effective way, other than through experience, for men to learn that those who have been trained in the symptom-complex of animal diseases are best qualified to deal with them. The empiric

who gives a drug in one case because he has seen it do good in another of apparently the same sort, acts upon the theory that similarity of superficial symptoms means similarity of lesions, which is perhaps as wild an hypothesis as could be invented. Animal husbandry has a great field in teaching owners how to keep their live stock well through proper care, nutrition and hygiene, but when these fail and disease appears, veterinarians are the best advisers. In Europe the people learned long ago that the treatment of animal diseases should be entrusted to veterinarians, which explains the large registration in veterinary colleges abroad.

INSTRUCTION

Considerable new equipment has been provided to improve the facilities for teaching. A modern refractometer, a polariscope and a microspectroscope have been added to the equipment in the Department of Physiology. The X-Ray machine, purchased last year, has proved to be of much assistance in diagnosis and this year an Alpine sun lamp has been added to the Department of Materia Medica and Small Animal Clinic for therapeutic purposes. The clinics have had a large increase in the number of cases, which has assured an abundance of material for practical teaching. The second story of the Surgical Hospital has been completed, which has increased materially the number of stalls for surgical cases. This additional space will make it possible to accommodate many more animals on which surgical operations of an instructive nature are desired, and which, because of the excellent work in the department, the owners seek to have done here.

The Department of Medicine, Obstetrics and Ambulatory Clinic has been organized in such a manner that one of the staff is available at all times to attend calls and to take with him one or more senior students. The valuable part of this clinic is that the cases are followed up until they have recovered or are examined post mortem to check up the diagnosis and treatment. This gives the student unusual opportunities to acquire knowledge of great practical value. A study of the report of the clinic will show that practically every disease of animals with which practitioners come in contact in this latitude is included. Splendid opportunities are afforded to study breeding diseases, especially of cattle, which have come to be a very important part of actual practice in this State. Evidence of the high opinion entertained by Europeans of the work that is being done in this department is found in the fact that a description of its investigations on genital diseases in cattle, in cooperation with Dr. W. L. Williams, published in the Cornell Veterinarian, was reprinted in The *Deut. tierärtzliche Wochenschrift,* January, 1926.

The department has the advantage of having a laboratory at its disposal. Its use teaches, in a very practical way, the necessity for accurately determined facts relative to the nature of the disorders encountered. Mention also should be made of the work which

is done by the department in editing and publishing the Cornell Veterinarian, a quarterly publication, on a self-supporting basis, for the benefit of veterinary practitioners. It is a medium of direct communication between the clinics and laboratories of the college and the practitioners of the State, thereby conveying new discoveries and results of research and clinical findings to the profession.

The instruction in Materia Medica, Therapeutics and Small Animal Diseases has been very satisfactory. The increased number of cases in the small animal clinic has made it unusually valuable for teaching and exceptionally difficult for the department to meet the demands. An additional assistant has been provided for next year. The efficient work in this clinic has, as in the other clinics, brought many animals to it from long distances. The indications now are that in a short time it will be necessary to enlarge the small animal hospital, as frequently every kennel is occupied and patients still coming. Too much emphasis cannot be placed on the opportunity and importance of the Small Animal Clinic in teaching materia medica and therapeutics.

The clinical departments are equipped with facilities and teachers to prepare students in surgery, medicine and obstetrics, and diseases of small animals, to perform the work expected of them by live stock owners. The appended table gives the number of cases treated in each clinic.

CLINIC	*Number of Cases*
Ambulatory and medical	6,261
Surgical and consulting	1,435
Small animal	1,774
Total	9,470

Exceptional opportunities for students in the upper classes to familiarize themselves thoroughly with important phases of veterinary work, in addition to the regular instruction given in connection with their subjects, are afforded in five distinct activities in the college; namely, post mortem examinations; laboratory diagnosis; the preparation of tuberculin and autogenous vaccines; poultry diseases; and the preparation of anti-hog-cholera serum. The volume of work in these subjects that was done this year is given in the appended table.

Post mortems	310
Diagnosis Laboratory—Number of blood specimens examined	4,783
Diagnosis Laboratory—Number of other specimens examined	1,321
Tuberculin made and distributed to veterinarians (doses)	49,219
Autogenous vaccines and bacterins (c.c.)	61,400
Mallein (c.c.)	10
Poultry specimens for diagnosis—Ithaca	1,385
Poultry specimens for diagnosis—Farmingdale	1,036
Anti-hog-cholera serum distributed (c.c)	208,440
Hog cholera virus for immunizing purposes (c.c.)	6,821

The diseases of poultry have come to be of much economic importance in the State. The industry has grown and with it the

losses from disease have been accelerated. Veterinarians are the people familiar with the nature of diseases who best can minimize these losses. This new responsibility is assumed cheerfully by many practitioners while others feel that they have not been trained in the maladies of fowls sufficiently to undertake their treatment. To overcome this objection for future graduates the amount of instruction in poultry diseases in the regular curriculum has been increased. The diagnoses of poultry diseases that are being made offer splendid opportunities in this work for interested students.

The demands on the part of poultrymen for assistance in the prevention and treatment of poultry diseases were so urgent that to aid practitioners a short course on poultry diseases was given especially for them. It was well attended and much interest was shown in the work. The instruction consisted largely in making diagnoses (an abundance of material was provided) and demonstrations.

A laboratory for the diagnosis of poultry diseases was organized early last July in cooperation with the State Institute of Applied Agriculture at Farmingdale, Long Island. A large amount of diagnosis work has been done there, as the report of Dr. Hendrickson, who is in charge, will show. In June a conference with the veterinarians of Long Island was held at Farmingdale in connection with poultry diseases. It was well attended and the veterinarians were much interested. Arrangements were made whereby they could receive, at stated times, technical instruction at the laboratory. It is of interest to note that the cause of the larger proportion of the troubles with poultry has been found to be improper feeding, poor sanitation and parasites, the correction of which falls naturally in the routine of practitioners. The destructive infectious diseases have been found not to be as common as was supposed.

Thorough teaching of horseshoeing to veterinary students has become a matter of increasing importance, as professional horseshoers are disappearing quite rapidly. The farriery is well equipped for this purpose and Professor Asmus is amply qualified to teach the subject. Veterinarians will be obliged to supervise more than heretofore the shoeing for lameness. The horse owners, especially in the country, are learning to nail on shoes, but as they are not familiar with the anatomy of the foot they are not prepared to do the work where special technical knowledge is required. During the year, Professar Asmus has cooperated with the Extension Department in the College of Agriculture, and at its request has held demonstrations in shoeing in thirty-six places, with a total attendance of 1222. This is a valuable service. It is hoped, notwithstanding the facts to the contrary, that this condition is temporary and that men enough will take up horseshoeing as a trade to provide the necessary service. The practice of working farm horses without shoes, advocated by some, is proving to be unsatisfactory.

LIBRARY

For many years the library of the college was crowded into a small room with inadequate funds for a librarian. Under such conditions, it was not used as a library should be by the student body. In 1924, the appropriation for a librarian was increased and in the fall of that year the new quarters in the South Wing of James Law Hall provided ample space. A full time librarian was employed who has given untiring service in classifying and cataloging the books, periodicals, bulletins and reprints which had accumulated, and assisting students in learning to use a library. This has made the library especially helpful, with the result that it is utilized much more than in the past. But the endowment does not provide income sufficient to purchase all the books and periodicals the college needs.

The library contains 7,625 volumes, of which 7,018 were purchased from the Flower fund and 607 were secured by gift or from state appropriations. It receives 113 periodicals, of which forty-eight are procured through exchange or by gift. The books and periodicals have been selected with much care, so that the library, while not large, possesses a most useful collection of literature on veterinary and closely allied sciences. A full time, efficient librarian has increased greatly the use of books and periodicals by students and it is anticipated that they will become a source of much assistance to the practitioners of the State.

VETERINARY CONFERENCE

The eighteenth annual conference for veterinarians was held January 14 and 15. The attendance was unusually large and keen interest was taken in the subject matter presented. Experience has shown that these short annual conferences, with programs consisting of subjects relating to the newer discoveries and practices in the science and art of veterinary medicine, are of great help to busy practitioners. Such men cannot leave their clients for long periods of study, but usually they can arrange to attend a short series of lectures and demonstrations. The amphitheatre with its moving picture machine, balopticon and space for practical demonstrations on living animals, has added much to the facilities for presenting papers and demonstrations.

The titles of the papers presented and the demonstrations made at the conference are appended.

Address of welcome ..... President Livingston Farrand
Growth of veterinary science and the application of new knowledge
V. A. Moore
A comparison of the blood of the normal male and two castrated goats ..... P. A. Fish and C. E. Hayden
A study of proprietary mineral mixtures and vitamin products in feeding of farm animals ..... L. A. Maynard
Department of Animal Husbandry, College of Agriculture
Poultry diseases on Long Island ..... J. M. Hendrickson
What kind of milk ordinance should we have in New York State? .. J. D. Brew
Dairy Department, College of Agriculture

Lantern slide demonstration of lymph glands.....G. S. Hopkins
The importance of bovine infection in man and the means of preventing it, such as milk pasteurization, tuberculin testing of cattle and vaccination of children and calves.....W. H. Park
Director of Research Laboratories, New York City Board of Health
Some phases of avian tuberculosis and their bearing on the general tuberculosis problem.....L. Van Es
University of Nebraska, Lincoln, Neb.
The "Hell" of it.....B. A. Pyrke
Commissioner of Farms and Markets, Albany, N. Y.
Arteriosclerosis in domesticated animals (lantern slides).....S. A. Goldberg
Military education, supplemented by the film, "Life O' Riley" (showing riding activities at the Cavalry School, Fort Riley, Kansas)
Capt. E. M. Curley

Demonstrations—
of the agglutination test for Bang abortion disease.....C. M. Carpenter
of the agglutination test for bacillary white diarrhea.....E. L. Brunett
of methods for examination of feces for parasites.....H. C. Stephenson
Hog cholera control, more especially on permanent immunization of young pigs.....M. Dorset
Chief of Biochemic Division, U. S. Bureau of Animal Industry
Washington, D. C.
Genital diseases of horses (lantern slides).....W. L. Williams
Treatment of diseases of the genital organs, including records for the last year.....C. H. Case, Akron, Ohio.
The agglutination test as an aid in the handling of the Bang abortion disease.....R. R. Birch and H. L. Gilman
Bacterium abortus in the udders of vaccinated and naturally infected cows.....C. M. Carpenter
The interpretation of clinical symptoms.....D. H. Udall
The production of anesthesia for various operations.....J. N. Frost
Some problems in small animal practice.....H. J. Milks
Education preparatory to veterinary medicine.....L. Van Es
A visit to Hippocrates and Galen.....Prof. S. H. Gage

Most of the papers presented were printed in the April number of the Cornell Veterinarian and a copy was available for each practitioner of the State. This was done to make it possible for those who were unable to attend to receive as much benefit as possible from the conference.

LABORATORY DIAGNOSIS

The application of scientific methods in the diagnosis of morbid conditions is emphasizing the necessity of laboratory service in the successful prosecution of veterinary medicine. Practitioners are utilizing the laboratory more extensively than heretofore. The assistance it gives by way of a rapid diagnosis makes possible the prompt enforcement of preventive measures that otherwise would be difficult or impossible to apply. The report of Dr. Carpenter on the work of the laboratory will show the variety and extent of its services.

The development of methods of greater precision in diagnosis has encouraged practitioners to utilize laboratories for many examinations that they themselves might make. In a former report attention was called to the importance of veterinarians equipping

themselves for the simpler examinations they were taught in their college course to make. Progress in veterinary medicine is being made as practitioners include, as a part of their procedure, the technical methods that have been perfected by laboratory and research workers and turned over to them for application. Advancement requires that the art of the profession should absorb as much of its science as possible. The college laboratories should be relieved from the examinations that practitioners can do, and which it would be in every way to their advantage to do, thereby enabling the laboratory men to do more research and to make those diagnoses that require facilities and experience possessed by the specialists. This is being done to a much greater extent than formerly.

RESEARCH

The statute establishing this college provides that one of its functions shall be to do research work in connection with animal diseases. The funds provided for this purpose have been inadequate to meet the needs. For that reason it has not been possible to initiate extended researches except in connection with the breeding diseases, for which a definite, but meagre, appropriation has been made. While there are very urgent demands for further knowledge of animal diseases, the needs for research in every branch of veterinary science are apparent. For this reason members of the faculty have undertaken such investigations as their time and limited funds allowed with the hope of strengthening as many as possible of the "weak links" in the chain of veterinary knowledge.

An important factor in the advancement of veterinary medicine is the application of new facts and methods relative to animal diseases. In recent years facilities for such researches have been better throughout the country and new knowledge has been accumulating faster than it has been applied. One of the problems of teachers in veterinary colleges is to accelerate the utilization of newly discovered truths. While progress in this respect appears to be slow, the facts indicate that it is more rapid than heretofore. The keen interest taken by many practitioners in the control of Bang abortion disease and in the prevention and treatment of poultry diseases is evidence of a scientific awakening in the profession. Cooperation is growing between laboratory workers and practitioners, whereby practical tests suggested by research are beng made. During the year this has been in evidence in connection with the use of autogenous vaccines and bacterins, sera tests for diagnosing Bang abortion disease and bacillary white diarrhea, the study of the blood in cases of milk fever and azoturia, and the adjusting of food in instances where deaths were occurring apparently from malnutrition. These cooperative studies are doing much to improve the practice of veterinary medicine.

The appendix will contain reports and papers on the following subjects:

The clinics, diagnosis laboratory and poultry disease investigation.
The interpretation of clinical symptoms.
Convulsions in dogs.
A comparison of the blood of a normal and two castrated billy goats.
The agglutination test as an aid in handling Bang abortion disease.
Bang abortion disease in cattle.
Technique of drawing blood samples from cattle for the agglutination test.
Immunizing young pigs against hog cholera.
The passage of *Bacterium abortum* through the digestive tract of cattle.
The value of placental examination for the diagnosis of infectious abortion of cattle.
Bacillary white diarrhea.
Directions for collecting blood samples for the agglutination test for infectious abortion.
The differentiation of *Bact. pullorum* (Rettger) and *Bact. sanguinarium* (Moore).
Undulant fever in man associated with bacteria indistinguishable from *Brucella abortus.*
Growth of veterinary science and the application of new knowledge.
Agglutinins for *Brucella abortus* in the blood serum of man.
A comparison of strains of *Brucella abortus* isolated from man with those from cattle.

I wish to call attention especially to the reports of the clinics. They show a total increase of 2,537 cases over those of the previous year. This emphasizes the opportunities here for the study of practical medicine and surgery.

The report of the diagnosis laboratory is interesting in that it shows a marked reduction in the number of cases of anthrax and rabies over those of some years ago. The large increase in the amount of work in the laboratory is due to the tests for infectious abortion and the preparation of autogenous bacterins and vaccines. There is a decrease in the quantity of tuberculin requested and the call for mallein has practically ceased. The reports of the diagnosis work in connection with poultry diseases, both at Ithaca and Farmingdale, are instructive in that they point out the diseases that are causing the larger amount of loss. Contrary to the general opinion, the specific infections are less serious than the general diseases, including malnutrition, parasites and insanitary environment. This shows that the ailments of poultry are to be dealt with in the same manner as those of other species. This will be helpful to veterinarians in poultry districts.

The studies that point out the apparent relationship that exists between the Bang abortion disease in cattle and certain fevers in man are of unusual interest. The discovery of an organism indistinguishable from *Brucella abortus* in the blood of persons suffering from an undulant fever raises important questions in connection with the production of pure milk for veterinarians and sanitarians to solve. The application of the agglutination test for the diagnosis of Bang abortion disease and bacillary white diarrhea has given to veterinarians methods of much practical value. Each report contains a contribution to veterinary science.

NEEDS OF THE COLLEGE

As this college represents the activities of the State in connection with the study of animal diseases, I wish again to emphasize the importance and great need of more liberal appropriations for their study and the construction of a laboratory better adapted to that purpose. The fact that millions of dollars are being expended annually in this State for eradicating bovine tuberculosis, which became established in this country in advance of veterinary service, is sufficient evidence that every reasonable precaution should be taken to prevent the development and spread of other diseases already here, which, if not controlled, will become an equally heavy burden in the not distant future.

The small number of veterinary students during the past few years has resulted in an inadequate number of trained veterinarians in the country. This condition is reflected in the opportunities that come to teachers in veterinary colleges. In order to keep competent and well prepared teachers, it is necessary that they receive adequate compensation. The needs of the college, therefore, consist in greater facilities and appropriations for the study of animal diseases and compensation for our teaching staff commensurate with their preparation and the work they are doing. Additional assistants are needed badly in the diagnosis laboratory and at least one more stenographer is required.

APPROPRIATIONS

The Legislature of 1926 appropriated for the maintenance of the college for the fiscal year 1926–27, $154,990. This includes an increase of $7,000 for the study of poultry diseases and $5,000 for initiating an investigation is connection with Johne's disease. There were no appropriations for new construction.

RECOMMENDATIONS

It is recommended that the trustees request of the Legislature of 1927 a sum sufficient for maintenance of the college, to increase the research work on animal diseases, to provide the necessary additional help, and to make just and deserved promotions. The specific amounts required for these purposes will be submitted in the budget requested for the fiscal year 1927–28.

PUBLICATIONS AND ADDRESSES OTHER THAN THOSE INCLUDED IN THE ANNUAL REPORT BY MEMBERS OF THE FACULTY

ASMUS, HENRY

Held 48 demonstrations in horseshoeing in eleven counties of the state for the Extension Department of the College of Agriculture.

BRUNETT, EARL L.

Poultry Diseases and Control Measures which may be Employed by Poultrymen. Address, Poultry Judging and Breeding School, Ithaca, N. Y., July, 1925.

Sanitary Measures in the Prevention of Poultry Diseases. Two-day field demonstration held on poultry farms in the vicinity of Rochester, N. Y., Sept., 1925.

The Importance of Disease Control and How Veterinarians can Cooperate with Poultrymen. University of Pennsylvania Veterinary Conference, January, 1925.

Six lectures and demonstrations on Poultry Diseases. Farmers' Week Program, February, 1926.

CARPENTER, C. M.

Undulant Fever in Man. Address, Society American Bacteriologists, December 28, 1925, Madison, Wis.

Report of Two Cases of Undulant Fever due to Basteria Indistinguishable from *Brucella abortus*. *Journal American Medical Association*, 1926.

Brucella abortus in the Udders of Vaccinated and Naturally Infected Cattle. *Cornell Veterinarian*, April, 1926.

The Agglutination Test for Infectious Abortion in Cattle. Address, New York State Veterinary Medical Society, June 29, 1926, Cortland, N. Y.

Brucella abortus Infection in Man. Address, Tompkins County Medical Society, June, 1926, Freeville, N. Y.

Brucella abortus in Market Milk. Address, Milk Distributors and Producers at Ithaca, N. Y.

FINCHER, M. G.

Arrested Development of the Muellerian Ducts Associated with Inbreeding. *Cornell Veterinarian*, January, 1926. Joint authorship with W. L. Williams.

FROST, J. N.

Diseases of the Udder and Teats. Missouri Valley Veterinary Medical Society, Omaha, Neb.

Surgical Diseases of the Genital Organs. Missouri Valley Veterinary Medical Society, Omaha, Neb.

Surgical Diseases of the Genital Organs. Minnesota State Veterinary Medical Society, St. Paul, Minn.

Laparotomy in Cattle. American Veterinary Medical Association, Portland, Ore.

Diseases of the Udder and Feet of Cattle. Vermont State Veterinary Medical Society, Burlington, Vt.

Anesthesia for Various Operations. Conference, New York State Veterinary College, January 15, 1925.

MILKS. H. J.

The Significance of Certain Prominent Symptoms in Dogs. Southern Tier Veterinary Medical Society, December, 1925.

Important Diseases of Dogs. Genesee Valley Veterinary Association.

Digestive Diseases of Dogs. Address at Ohio State Conference. To be published in their *Alumni Journal*.

Some Diseases of Dogs. New York State Veterinary College Conference, January 15, 1925.

MOORE, VERANUS A.

The Value of Animal Experimentation. *Cornell Sun*, March 26, 1926.

Anthrax. Nelson's Looseleaf Medicine.

The Aim of City Milk Control. Address to Dairymen, Farmers' Week Program, College of Agriculture, February, 1926.

Two lectures on Meat and Dairy Inspection. Massachusetts Institute of Technology, April 2nd and 3rd, 1926.

Two lectures, Ray Brook, April 24, 1926.

a. Tuberculosis of Animals and Its Relation to that of Man.

b. The Control of Bovine Tuberculosis.

The Nature and Differentiation of Poultry Diseases. Long Island Poultry Association, Farmingdale, L. I., June 24, 1926.

Veterinary Science as an Economic Factor. Address, Philadelphia Rotary Club. *Cornell Veterinarian*, January, 1926.

The Significance of Crises in the Veterinary Profession. *Cornell Veterinarian*, October, 1925.

FINANCIAL STATEMENT

REPORT OF THE CIRCULATING FUNDS OF THE NEW YORK STATE VETERINARY COLLEGE AT CORNELL UNIVERSITY, JULY 1, 1925, TO JUNE 30, 1926.

Balance July 1, 1925		$6,397 39
Receipts 1925–1926:		
Tuition	$1,435 00	
Laboratory fees	2,147 45	
Clinics	10,097 27	
Horseshoeing	1,818 20	
Tuberculin and vaccines	2,005 30	
Blood tests	170 80	
Hog cholera serum	3,023 05	
Animals sold	3,523 70	
Apparatus and supplies	422 03	
Rent	141 00	
Refunds and miscellaneous	90 54	
		24,874 34
Disbursements 1925–1926:		
Summer work	$3,175 00	
Night and Sunday work	641 00	
Overtime	602 80	
Outside labor	239 15	
Laboratory work	272 40	
Clerical work	425 03	
Lectures and drawing	97 00	
Equipment	1,403 88	
Supplies	4,504 20	
Printing and advertising	898 15	
Communication	693 93	
Traveling and hotel	1,008 18	
Automobile upkeep	3,302 12	
Repairs	78 59	
Fuel, etc.	695 06	
Laundry	353 67	
Animals	340 35	
Taxes and insurance	407 02	
Miscellaneous	31 73	
	$19,169 26	
Balance July 1, 1926	12,102 47	
	$31,271 73	$31,271 73

EXPENDITURE OF THE MAINTENANCE FUNDS FOR THE NEW YORK STATE VETERINARY COLLEGE FOR THE YEAR 1925–1926 — CHAP. 181/1925.

Administration, salaries	$12,660 00
Teaching and research, salaries	69,690 00
Care of plant, wages	13,020 00
Temporary labor	2,400 00
Fuel, heat, light, power, water	8,500 00
Printing	1,966 68
Equipment, supplies, materials	7,497 13
Traveling expenses	452 84
Communication	1,250 00
Repairs	1,782 08
Investigation of animal diseases	17,499 35
	$136,718 08
Balance to revert to State	301 92
Appropriation	$137,020 00

EXPENDITURE OF THE SPECIAL FUNDS FOR THE NEW YORK STATE VETERINARY COLLEGE FOR THE COLLEGE YEAR 1925–1926.

Balances July 1, 1925:			
Addition to laboratory building (Chapter 140, 1924-5)			$21,898 37
Equipment, furniture, etc. (Chapter 225, 1923-3)			160 15
Addition to serum laboratory (Chapter 225, 1923-3)			13 82
Repair of parapet (Chapter 225, 1923–3)			1,140 31
Surgical building, second story (Chapter 140, 1924-3)			36,933 01
Expenditures 1925–1926:			
Addition to laboratory building	$4,348 78		
Equipment, etc.	155 57		
Surgical building	31,223 13		
		$35,727 48	
Reverted to State:			
Addition to serum laboratory	$13 82		
Repair of parapet	1,140 31		
		1,154 31	
Balances July 1, 1926:			
Addition to lab. bldg	$17,549 59		
Equipment, etc.	4 58		
Surgical building	5,709 88		
		23,264 05	
		$60,145 66	$60,145 66

As per itemized vouchers approved by the State Comptroller.

H. H. HAIGHT,
Clerk.

The appendix, which contains the reports of the clinics, the poultry disease investigations and those of research work, is an integral part of this report.

The success that has been attained in both teaching and research has been possible because of the hearty cooperation and untiring efforts of the members of the faculty.

Respectfully submitted,

V. A. MOORE,

Dean New York State Veterinary College at Cornell University.

APPENDIX

Containing Reports of the Clinics and Research

REPORT OF THE AMBULATORY CLINIC

From *July* 1, 1925, to *June* 30, 1926

D. H. UDALL, E. R. CUSHING, and M. G. FINCHER

Department of Veterinary Medicine, Obstetrics, and Ambulatory Clinic

The attached report of the animals treated in the Ambulatory Clinic during the year ending June 30, 1926, shows a total of 6,261 cases, divided as follows:

Cattle	4,792
Swine	690
Horses	470
Sheep	309
Total number of animals treated or examined	6,261

This report shows an increase in the number of animals treated and examined, including all species. Much of the increase is due to demands for physical examinations and tuberculin tests in cattle, but in addition to these the practice in general has continued to grow.

The ambulatory clinic is of inestimable value as a source of information to both students and teachers. The volume is sufficient to provide experience in the control of the most important diseases of this State, and in the diagnosis and treatment of the vast majority of affections met with in general practice. It affords an opportunity to test new methods of treatment under field conditions. Some of these now under observation will be reported upon when the final results are obtained.

Cases Treated in the Ambulatory Clinic

NAME OF THE DISEASE	Horses	Cattle	Sheep	Swine	Recovered	Improved	Not improved	Killed or died	Undetermined
1. Infectious Diseases									
Actinomycosis	....	3	....	....	1	....	2	....	.....
Coccidiosis	....	1	....	....	1	....	....	....	.....
Cow pox	....	1	....	....	1	....	....	....	.....
Diseases of the new-born	....	15	....	....	7	....	....	6	2
Hemorrhagic septicemia	....	8	1	....	1	....	....	8	.....
Influenza	1	....	....	....	....	....	....	1	.....
Malignant head catarrh	....	3	....	....	....	....	....	2	1
Necrobacillosis	1	2	....	....	....	....	....	2	1
Purpura hemorrhagica	1	....	....	....	1	....	....	....	.....
Septicemia	....	1	....	....	....	....	....	1	.....
Strangles	5	....	....	....	5	....	....	....	.....
Tetanus	3	....	....	....	....	....	....	3	.....
2. Diseases of the Circulatory System									
Azoturia	8	....	....	....	4	....	....	4	.....
Lymphangitis	13	....	....	....	10	....	....	....	3
3. Diseases of the Respiratory System									
Abscess, pulmonary	....	2	1	....	....	....	....	3	.....
Bronchitis	....	1	....	....	....	....	....	....	1

NAME OF THE DISEASE	Horses	Cattle	Sheep	Swine	Recovered	Improved	Not improved	Killed or died	Undetermined
Congestion, pulmonary	1	....	1	....	1	....	....	1	.....
Coryza	3	....	....	....	3	....	....	....	.....
Heaves	8	....	....	....	1	1	1	....	5
Laryngitis	3	....	....	....	2	1	....	....	.....
Pharyngitis	1	....	....	....	1	....	....	....	.....
Pneumonia	....	17	2	....	1	....	....	14	4
Snuffles	....	1	....	....	....	1	....	....	.....
4. DISEASES OF THE DIGESTIVE SYSTEM									
Cancer, intestinal	1	....	....	....	....	....	....	1	.....
Catarrh, gastro-intestinal	14	17	....	....	31	....	....	....	.....
Choke	2	1	....	....	3	....	....	....	.....
Dysentery, winter	....	96	....	....	96	....	....	....	.....
Enteritis	4	18	1	3	10	....	....	12	4
Enteritis, necrobacillosis	....	1	....	....	....	....	....	1	.....
Incarceration	1	....	....	....	....	....	....	1	.....
Indigestion	73	75	....	2	130	....	....	3	17
Parasitism, ascariasis	1	....	....	....	1	....	....	....	.....
Parasitism, gastrophilus nasalis	1	....	....	....	....	....	....	1	.....
Parasitism, stomach-worm disease	....	....	32	....	3	....	7	1	21
Parasitism, strongylidosis	11	....	....	....	10	....	....	1	.....
Peritonitis	1	1	....	....	....	....	....	2	.....
Prolapse of rectum	1	....	....	1	2	....	....	....	.....
Rupture, duodenum	1	....	....	....	....	....	....	1	.....
Rupture, gastric	4	....	....	1	....	....	....	5	.....
Rupture, rumen	....	1	....	....	....	....	....	1	.....
Stomatitis	1	....	....	....	1	....	....	....	.....
Teeth, sharp, etc	55	....	....	....	....	....	....	....	.....
Traumatic gastritis	40	....	....	....	....	11	....	19	10
5. DISEASES OF THE GENITO-URINARY SYSTEM									
Aberration, genitals	....	2	....	....	1	....	....	1	.....
Abortion	1	108	....	2	78	2	....	9	22
Balanitis	5	....	....	....	4	1	....	....	.....
Cervicitis	....	8	....	....	4	....	1	2	1
Cystic ovaries	....	5	....	....	2	....	1	....	2
Dystocia	1	41	....	7	38	....	....	10	1
Endometritis	....	2	....	....	....	....	....	....	2
Examination of male genitals	....	19	....	....	....	....	....	2	3
Examination of female genitals	....	50	....	....	....	....	....	....	.....
Hemorrhage, uterine artery, after calving	....	1	....	....	....	....	....	1	.....
Hydrops amnion	....	1	....	....	1	....	....	....	.....
Mastitis	....	185	....	....	31	15	14	6	119
Metritis	....	78	....	....	56	1	1	15	5
Milk, bitter	....	1	....	....	1	....	....	....	.....
Milk fever	....	42	....	....	37	....	....	....	5
Monstrosity	....	2	....	....	2	....	....	....	.....
Mummified fetus	....	4	....	....	3	....	....	....	1
Navel ill	....	....	4	....	....	....	....	....	4
Nephritis	....	1	....	....	....	....	....	....	1
Oedema, udder	....	1	....	....	1	....	....	....	.....
Ovaritis	....	5	....	....	....	....	....	3	2
Paralysis, postpartum	....	4	....	....	2	....	....	2	.....
Pregnancy examination	....	61	....	....	....	....	....	....	.....
Prolapse uterus	....	19	....	....	13	....	....	6	.....
Prolapse vagina	....	11	....	....	11	....	....	....	.....
Pyelonephritis	....	5	....	....	....	....	....	5	.....
Retained placenta	....	124	....	....	87	2	5	5	25
Rupture of uterus	....	1	....	....	....	....	....	1	.....
Sterility	....	83	....	....	29	1	7	10	36
Teats, diseases and injuries	....	73	....	....	31	....	....	....	42
Torsion of uterus	....	10	....	....	9	....	....	1	.....
Vaginitis	....	18	....	....	16	....	....	1	1
6. DISEASES OF THE LIVER									
Abscess	....	2	....	....	....	....	....	....	.....
Cholecystitis	....	....	1	....	....	....	....	1	.....
Cirrhosis	1	....	....	....	....	....	....	....	.....
Necrosis	....	1	....	....	....	....	....	....	.....

NAME OF THE DISEASE	Horses	Cattle	Sheep	Swine	Recovered	Improved	Not improved	Killed or died	Undetermined
7. DISEASES OF THE SKIN									
Dermatitis	8	6	....	....	14	....	....	....	.....
Lice	....	1	....	....	....	....	....	....	.....
Ringworm	....	1	....	....	....	....	....	....	.....
Urticaria	....	3	....	....	3	....	....	....	.....
8. DISEASES OF THE NERVOUS SYSTEM									
Dummies	1	....	....	....	....	....	1	....	.....
Encephalitis	....	2	....	....	....	....	....	2	.....
Meningitis	....	3	....	....	1	....	....	2	.....
Paralysis	....	3	4	....	2	....	....	5	.....
9. DISEASES OF THE EYE									
Conjunctivitis	1	....	....	....	1	....	....	....	.....
Foreign bodies	1	1	....	....	2	....	....	....	.....
Keratitis	....	1	....	....	....	....	....	....	1
Periodic ophthalmia	17	....	....	....	2	10	2	....	3
10. EXTERNAL DISEASES									
Abrasion	2	....	....	....	2	....	....	....	....
Abscess	11	19	....	2	11	3	....	....	18
Arthritis	4	2	1	....	2	....	....	1	4
Atrophy	5	1	....	....	....	3	....	....	3
Bursitis	3	....	....	....	3	....	....	....	.....
Castration	11	1	126	100	237	....	....	1	.....
Contusion	6	6	....	....	12	....	....	....	.....
Dehorning	....	287	....	....	....	....	....	....	.....
Dehorning hemorrhage	....	3	....	....	3	....	....	....	.....
Dehorning sinusitis	....	4	....	....	4	....	....	....	.....
Docking tails	....	....	125	....	....	....	....	....	.....
Fistula	4	1	....	....	1	....	....	....	4
Fracture, humerus	1	1	....	....	....	....	....	2	.....
Fracture, ilium	1	1	....	....	....	....	....	2	.....
Fracture, pubis	1	1	....	....	....	....	....	2	.....
Fracture, rib	1	1	....	....	2	....	....	....	.....
Fracture, tibia	3	1	....	....	....	....	....	4	.....
Hematoma	9	4	....	....	12	....	....	....	1
Hernia	....	2	....	1	....	1	2	....	.....
Hygroma	....	5	....	....	5	....	....	....	.....
Laminitis	8	....	....	....	4	....	1	....	3
Phlegmon	....	6	....	....	6	....	....	....	.....
Pododermatitis	18	....	....	....	13	....	....	....	5
Pododermatitis, "fouls"	....	92	....	....	88	1	1	....	2
Poll evil	1	....	....	....	....	....	....	....	1
Quittor	1	....	....	....	1	....	....	....	.....
Ringbone	1	....	....	....	....	1	....	....	.....
Sprain	6	2	....	....	6	....	....	....	2
Strain	....	2	....	....	....	....	....	....	2
Tendinitis	7	1	....	....	6	....	....	....	2
Tendovaginitis	5	4	....	....	7	1	....	....	1
Thrush	5	....	....	....	5	....	....	....	.....
Wounds	56	19	....	1	71	1	....	4	.....
11. POISONING									
Belladonna	....	1	....	....	1	....	....	....	.....
Iron sulphate	....	....	10	....	....	....	....	10	.....
Lead	....	2	....	....	....	....	....	2	.....
Magnesium sulphate	....	1	....	....	....	....	....	1	.....
Salt	....	11	....	....	11	....	....	....	.....
12. MISCELLANEOUS									
Cast	5	....	....	....	2	....	....	3	.....
Physical examination	....	1,300	....	....	....	....	....	....	.....
Ringing bulls	....	5	....	....	....	....	....	....	.....
Tuberculin test	....	1,600	....	....	....	....	....	42	.....
Vaccination, abortion	....	11	....	....	....	....	....	....	.....
Vaccination, hemorrhagic septicemia	....	60	....	....	....	....	....	....	.....
Vaccination, hog cholera	....	....	....	570	....	....	....	....	.....
Vaccination, mastitis	....	61	....	....	....	....	....	....	.....
Vaccination, metritis	....	20	....	....	....	....	....	....	.....
Total	470	4,792	309	690	....	....	....	....	.....

LABORATORY EXAMINATIONS FOR AMBULATORY CLINIC

HERBERT L. GILMAN

Bacterins:	
Bact. abortum	22
Mastitis	71
Blood agglutinated for Bact. abortum infection	662
Blood cultures	5
Cultures:	
Feces	1
Miscellaneous organs	14
Placenta or uterine exudate	20
Pus	10
Vaginal mucus	13
Feces examination for parasitic ova	11
Fetuses posted and cultured	28
Genital tracts cultured and described:	
Female	28
Male	3
Guinea pigs injected and examined for Bact. abortum:	
Fetuses	49
Genital tracts	21
Milk	276
Miscellaneous organs and glands	11
Placentas	98
Uterine exudates	129
Milk count, bacterial	2
Milk injected guinea pigs for tuberculosis	66
Milk samples cultured	118
Pus, examination for actinomycosis	5
Pus, suspected tuberculosis. Smears and inoculation	4
Sections, tissue	14
Semen:	
Cultured	9
Guinea pig inoculation	27
Stain and differential count	22
Skin scrapings for parasites	4
Smears, placenta. For Bact. abortum	13
Sputum samples, inoculation for tuberculosis	396
Vaccines, mastitis	75

REPORT OF THE SURGICAL AND CONSULTING CLINIC

July 1, 1925, to *June* 30, 1926

J. N. FROST and H. B. McMURRAY

NAME OF THE DISEASE	Horses	Cattle	Sheep	Swine	Goats	Recovered	Improved	Not improved	Killed or died	Undetermined
Abortion	5	6	...	...	...	9	...	...	...	2
Abortion test	...	129	...	...	...	...	...	...	...	....
Abscess	7	27	12	1	...	47	...	...	...	....
Amputation of teat	...	1	...	...	...	1	...	...	...	....
Arthritis	...	1	...	...	...	1	...	...	...	....
Azoturia	3	...	...	...	...	3	...	...	...	....
Balanitis	5	...	...	...	...	5	...	...	...	....
Bronchitis	3	9	...	...	...	12	...	...	...	....
Bursattee	2	...	...	...	...	...	2	...	...	....
Bursitis	2	9	...	...	...	11	...	...	...	....
Canker	4	...	...	...	...	3	...	1	...	....
Castration	7	1	1	10	1	20	...	...	...	....
Cervicitis	...	1	...	...	...	...	1	...	...	....
Choke	...	1	...	...	...	1	...	...	...	....
Chorea	...	...	1	...	...	...	1	...	...	....
Claws trimmed	...	31	...	...	...	...	...	...	...	....
Conjunctivitis	1	...	...	...	...	1	...	...	...	....
Contracted heels	3	...	...	...	...	3	...	...	...	....
Contusions	12	1	...	...	...	13	...	...	...	....
Cow pox	...	6	...	...	...	6	...	...	...	....
Cryptorchid	1	...	...	2	...	3	...	...	...	....
Debility	1	3	...	...	...	3	1	...	...	....
Dehorning	...	2	...	...	...	2	...	...	...	....
Dermatitis	5	7	...	...	...	12	...	...	...	....
Diarrhea	6	51	...	...	...	57	...	...	...	....
Dystocia	...	15	6	...	...	21	...	...	...	....
Eczema	1	...	...	...	...	1	...	...	...	....
Enteritis	...	1	...	...	...	...	...	...	1	....
Eversion uterus	...	1	...	...	...	1	...	...	...	....
Eversion vagina	...	1	...	...	...	1	...	...	...	....
Examination, pregnancy	...	57	...	...	...	...	...	...	...	....
Fistula anal	1	...	...	...	...	1	...	...	...	....
Fistula ear	1	...	...	...	...	1	...	...	...	....
Fistula tooth	2	...	...	...	...	2	...	...	...	....
Fistula withers	7	...	...	...	...	7	...	...	...	....
Foot rot	...	43	1	...	...	44	...	...	...	....
Fracture metatarsus	...	1	...	...	...	1	...	...	...	....
Fracture pelvis	1	...	...	...	...	...	...	...	1	....
Fracture vertebra	...	...	1	...	...	...	...	...	1	....
Gastritis	...	1	...	...	...	...	...	...	1	....
Glossitis	...	...	1	...	...	1	...	...	...	....
Hair root infection	1	...	...	...	...	1	...	...	...	....
Heaves	4	...	...	...	...	4	...	...	...	....
Hematome	3	11	...	...	...	14	...	...	...	....
Hemorrhagic septicemia vaccination	...	46	...	...	...	...	...	...	...	....
Hernia, scrotal	...	...	1	13	...	14	...	...	...	....
Hernia, umbilical	1	...	2	1	...	3	...	1	...	....
Hygroma	2	...	...	...	...	2	...	...	...	....
Impaction	...	6	1	...	...	6	...	...	1	....
Indigestion	12	24	1	...	...	35	...	...	2	....
Influenza	1	...	...	...	...	1	...	...	...	....
Keloid	1	...	...	...	...	1	...	...	...	....
Keratitis	1	...	...	...	...	1	...	...	...	....
Lip-leg ulceration	...	...	130	...	...	130	...	...	...	....
Lung worms	...	...	1	...	...	...	...	...	1	....
Lymphangitis	1	...	...	...	...	1	...	...	...	....
Mastitis	...	25	...	...	...	23	2	...	...	....
Metritis	...	30	1	...	...	27	...	...	4	....
Navicular disease	1	...	...	...	...	...	1	...	...	....
Necrophorus infection	1	5	...	...	...	6	...	...	...	....
Necrosis of lateral cartilage	2	...	...	...	...	2	...	...	...	....
Omarthritis	1	...	...	...	...	1	...	...	...	....
Open joint	1	...	...	...	...	...	...	...	1	....
Open tendon sheath	3	1	...	...	...	3	...	...	1	....
Ophthalmia	12	...	23	...	...	33	2	...	...	....
Otitis media	...	...	2	1	...	2	...	...	1	....
Paralysis obturator	...	...	...	1	...	1	...	...	...	....

NAME OF THE DISEASE	Horse	Cattle	Sheep	Swine	Goats	Recovered	Improved	Not improved	Killed or died	Undetermined
Parasites	11	...	5	...	...	15	...	...	1	....
Parturient paresis	...	4	...	...	...	4	...	...	...	....
Parturition	3	13	...	...	...	...	...	...	...	....
Pericarditis	...	1	...	...	...	...	...	...	1	....
Pharyngitis	1	...	...	...	...	1	...	...	...	....
Pneumonia	1	8	4	...	...	10	...	...	3	....
Pododermatitis	3	...	...	...	...	3	...	...	...	....
Poll evil	5	...	...	...	...	5	...	...	...	....
Quarter crack	3	...	...	...	...	3	...	...	...	....
Rectum prolapse	...	...	...	3	...	3	...	...	...	....
Resection of flexor pedis tendon	1	...	...	...	...	1	...	...	...	....
Retained placenta	2	29	1	...	...	31	...	...	1	....
Retention of urine	1	...	...	...	...	1	...	...	...	....
Ring bone	4	...	...	...	...	...	3	1	...	....
Roarer	7	...	...	...	...	7	...	...	...	....
Septicemia	2	6	1	...	...	5	...	...	4	....
Sesamoiditis	1	...	...	...	...	...	1	...	...	....
Shoe boil	1	...	...	...	...	1	...	...	...	....
Side bone	3	...	...	...	...	...	3	...	...	....
Sinking of the os pedis	3	...	...	...	...	...	3	...	...	....
Sitfast	2	...	...	...	...	2	...	...	...	....
Spavin	3	...	...	...	...	...	3	...	...	....
Spaying	5	...	...	...	...	5	...	...	...	....
Sterility	3	15	...	...	...	13	3	...	...	2
Stomatitis	...	1	9	1	...	10	...	...	1	....
Streptococcus bacterin injection	12	10	...	...	...	...	...	...	...	....
String halt	6	...	...	...	...	...	6	...	...	....
Teat, fistula of	...	1	...	...	...	1	...	...	...	....
Teat, stenosis of	...	1	...	...	...	1	...	...	...	....
Teeth, diseased	5	...	...	...	...	...	...	...	...	....
Teeth, examination	16	...	...	...	...	...	...	...	...	....
Teeth, long	9	...	...	...	...	...	...	...	...	....
Teeth, sharp	14	...	...	...	...	...	...	...	...	....
Teeth, split	3	...	...	...	...	...	...	...	...	....
Tendinitis	9	...	...	...	...	9	...	...	...	....
Tendovaginitis	1	...	...	...	...	1	...	...	...	....
Tetanus	2	...	...	...	...	...	...	...	2	....
Thrush	3	...	...	...	...	2	...	...	1	....
Torsion, uterus	...	1	...	...	...	1	...	...	...	....
Traumatic gastritis	...	6	...	...	...	4	...	...	2	....
Trochanteric lameness	1	...	...	...	...	1	...	...	...	....
Tuberculin test	...	103	...	...	...	...	...	...	...	....
Tumor, brain	1	...	...	...	...	...	...	...	1	....
Tumor, horn	8	...	...	...	...	8	...	...	...	....
Tumor, malignant	2	...	...	...	...	...	...	...	2	....
Tumor, non-malignant	1	...	...	...	...	1	...	...	...	....
Tumor, shoulder	6	...	...	...	...	6	...	...	...	....
Tumor, udder	...	...	...	1	...	1	...	...	...	....
Umbilical infection	6	7	...	...	...	12	...	...	1	....
Urethrotomy	1	...	...	...	...	1	...	...	...	....
Urinary calculi	1	...	3	...	...	3	...	...	1	....
Uterus ruptured	...	1	...	...	...	...	...	...	1	....
Vaginitis necrotic	...	12	...	...	...	10	...	...	2	....
White scours	...	4	...	...	...	3	...	...	1	....
Wound calk	12	...	...	...	...	12	...	...	...	....
Wound fistula	1	...	...	...	...	...	...	1	...	....
Wound miscellaneous	73	6	...	...	...	79	...	...	...	....
Wound nail	5	...	...	...	...	5	...	...	...	....
Miscellaneous cases:										
Bull ring in nose	...	2	...	...	...	...	...	...	...	....
Infected hock	...	13	...	...	...	10	3	...	...	....
Meconium retained	3	...	...	...	...	2	...	...	1	....

Total horses	394
Total cattle	798
Total sheep	208
Total swine	34
Total goats	1
Counted twice	30
Total cases treated	1,465
Grand total	1,435

REPORT OF THE SMALL ANIMAL CLINIC

July 1, 1925, *to June* 30, 1926

H. J. MILKS and H. C. STEPHENSON

Department of Materia Medica and Small Animal Clinic

The number of cases treated in the Small Animal Clinic was larger than for 1924–25. There were a few more canine and a few less feline patients, the increase in the former a little more than offsetting the decrease in the latter.

The list of cases includes the diseases ordinarily met in a small animal practice plus a few of more than ordinary interest.

CASES TREATED IN THE SMALL ANIMAL CLINIC

NAME OF DISEASE	Dogs	Cats	Recovered	Improved	Not improved	Died	Killed	Undetermined
1. INFECTIOUS DISEASES								
Distemper	155	....	85	....	....	53	14	3
Infectious gastroenteritis	....	29	1	....	....	28	....	.....
Infectious hemorrhagic gastroenteritis	1	....	....	....	....	1	....	.....
2. DISEASES OF THE CIRCULATORY SYSTEM								
Endocarditis	2	....	1	....	1	....	....	.....
3. DISEASES OF THE RESPIRATORY SYSTEM								
Bronchitis	4	8	7	....	1	2	2	.....
Coryza	10	13	22	1	....	....	....	.....
Empyema	2	1	....	....	....	3	....	.....
Laryngitis	21	3	24	....	....	....	....	.....
Pleuritis	....	1	....	....	....	1	....	.....
Pneumonia	1	2	....	....	....	3	....	.....
4. DISEASES OF THE NERVOUS SYSTEM								
Chorea	7	....	2	4	....	....	1	.....
Convulsions	26	....	20	....	....	6	....	.....
Paralysis, posterior	6	....	....	....	....	4	2	.....
Paralysis, radial	2	....	....	....	2	....	....	.....
Paralysis, vestibular	....	2	....	....	....	2	....	.....
5. DISEASES OF THE EYE								
Amaurosis	2	....	....	....	....	....	1	1
Cataract	2	....	....	....	2	....	....	.....
Conjunctivitis, catarrhal	12	....	10	2	....	....	....	.....
Conjunctivitis, follicular	4	....	4	....	....	....	....	.....
Eczema of eyelids	5	....	5	....	....	....	....	.....
Entropion	5	....	5	....	....	....	....	.....
Eyeball, enucleation of	1	....	1	....	....	....	....	.....
Foreign body in eye	1	....	1	....	....	....	....	.....
Hordoleum	1	....	1	....	....	....	....	.....
Iritis	1	....	1	....	....	....	....	.....
Keratitis, superficial	7	....	6	1	....	....	....	.....
Keratitis, traumatic	3	....	3	....	....	....	....	.....
Keratitis, ulcerative	14	1	13	1	....	1	....	.....
Membrana nictitans, removal of protruding	3	....	3	....	....	....	....	.....
Orbital gland, removal of tumefied	9	....	9	....	....	....	....	.....
Pannus	1	....	....	1	....	....	....	.....

NAME OF DISEASE	Dogs	Cats	Recovered	Improved	Not improved	Died	Killed	Undetermined
6. DISEASES OF THE GENITO-URINARY SYSTEM								
Balanitis	1	....	1	....	....	....	....	.....
Caesarian section	5	....	4	....	....	1	....	.....
Calculi, cystic	1	....	....	....	....	1	....	.....
Calculi, urethral	1	....	1	....	....	....	....	.....
Castration	*7	69	73	....	....	1	....	.....
Cystitis, hemorrhagic	1	2	1	....	....	1	1	.....
Dystocia	8	....	6	....	....	1	1	.....
Hypospadia	1	....	1	....	....	....	....	.....
Incontinence	2	....	1	1	....	....	....	.....
Mastitis	2	1	3	....	....	....	....	.....
Metritis	3	....	2	....	....	1	....	.....
Oestrum	1	1	....	....	....	....	....	.....
Oophorectomy	308	112	415	....	....	5	....	.....
Orchitis, suppurative	1	....	....	....	....	1	....	.....
Penis, wound of	1	1	1	....	....	....	1	.....
Urethra, stricture of	1	....	1	....	....	....	....	.....
7. DISEASES OF THE DIGESTIVE SYSTEM								
Choke	2	....	2	....	....	....	....	.....
Constipation	5	1	6	....	....	....	....	.....
Constipation, false	2	....	2	....	....	....	....	.....
Diarrhea	4	1	5	....	....	....	....	.....
Foreign body in mouth	1	....	1	....	....	....	....	.....
Gastritis	37	3	38	1	....	1	....	.....
Gastroenteritis	7	1	1	....	....	7	....	.....
Icterus	1	....	....	....	....	1	....	.....
Indigestion	29	5	33	....	....	1	....	.....
Obstruction of small intestine	2	....	....	....	....	2	....	.....
Pharyngitis	1	....	1	....	....	....	....	.....
Rectum, prolapse of	2	....	2	....	....	....	....	.....
Stomach, acute dilatation of	1	....	1	....	....	....	....	.....
Stomatitis, ulcerative	2	....	1	....	....	1	....	.....
Worms, hook	†38	....	31	....	....	7	....	.....
Worms, round	37	11	48	....	....	....	....	.....
Worms, tape	16	....	16	....	....	....	....	.....
Worms, whip	7	....	7	....	....	....	....	.....
8. DISEASES OF THE SKIN								
Eczema	70	4	70	4	....	....	....	.....
Fleas	8	15	....	....	....	....	....	.....
Furunculosis	6	....	3	2	1	....	....	.....
Lice	8	2	....	....	....	....	....	.....
Mange, follicular	46	....	31	5	3	2	5	.....
Mange, otodectic	25	6	30	1	....	....	....	.....
Mange, sarcoptic	25	36	47	1	....	10	3	.....
Ringworm	5	1	6	....	....	....	....	.....
Urticaria	2	....	2	....	....	....	....	.....
9. EXTERNAL DISEASES								
Abscess	12	5	17	....	....	....	....	.....
Amputation of dew claws	10	....	10	....	....	....	....	.....
Amputation of foot	1	....	....	....	....	1	....	.....
Amputation of leg	‡5	....	4	....	....	1	....	.....
Amputation of tail	34	1	35	....	....	....	....	.....
Anal sacs, impaction of	10	....	10	....	....	....	....	.....
Ankylosis of elbow	2	....	1	....	1	....	....	.....
Ascites	4	....	....	....	1	1	2	.....
Atresia, rectum	....	1	....	....	....	....	1	.....
Carcinoma	9	3	8	....	....	....	4	.....
Caries	....	1	....	....	....	....	1	.....
Concussion of brain	....	1	....	....	....	1	....	.....
Concussion of spine	1	....	....	....	....	....	1	.....
Cuterebra	§3	1	4	....	....	....	....	.....
Cysts, interdigital	4	....	4	....	....	....	....	.....
Cysts, on face	1	....	1	....	....	....	....	.....
Deafness	1	....	....	....	1	....	....	.....
Diaphragm, rupture of	1	....	....	....	....	1	....	.....

*1 Coyote. †2 Foxes. ‡2 Raccoons. §1 Rabbit.

NAME OF DISEASE	Dogs	Cats	Recovered	Improved	Not improved	Died	Killed	Undetermined
Erosion of atalanto axial	1	....	....	....	....	....	1	.....
Fracture of femur	10	....	8	....	....	1	1	.....
Fracture of humerus	2	....	2	....	....	....	....	.....
Fracture of mandible	1	....	....	1	....	....	....	.....
Fracture, compound of metatarsi	2	1	1	....	....	....	2	.....
Fracture of pelvis	2	....	2	....	....	....	....	.....
Fracture of radius and ulna	3	....	2	....	....	....	1	.....
Fracture of spine	2	....	....	....	....	....	2	.....
Fracture of tail	1	....	1	....	....	....	....	.....
Fracture of tibia	1	....	1	....	....	....	....	.....
Fracture of tibia and fibula	1	....	1	....	....	....	....	.....
Fracture, comp., of tibia and fibula	3	†2	2	....	....	....	3	.....
Fracture of ulna	2	....	2	....	....	....	....	.....
Hernia, inguinal	1	....	1	....	....	....	....	.....
Hernia, umbilical	10	....	9	....	....	1	....	.....
Injuries	7	2	9	....	....	....	....	.....
Injuries, internal	3	3	3	....	....	3	....	.....
Lameness	4	1	5	....	....	....	....	.....
Liver, enlarged	1	....	....	....	....	....	....	1
Luxation of elbow	2	....	....	1	1	....	....	.....
Luxation of hip	2	....	2	....	....	....	....	.....
Luxation of patella	2	....	2	....	....	....	....	.....
Needle in neck	1	....	1	....	....	....	....	.....
Osteosarcoma	1	....	....	....	....	....	1	.....
Otorrhea, catarrhal	6	....	5	....	....	....	1	.....
Papilloma	9	....	8	....	....	....	1	.....
Rupture of flexor tendons	2	....	1	....	1	....	....	.....
Rupture of heart	1	....	....	....	....	1	....	.....
Rupture, tendoachilles	1	1	1	....	1	....	....	.....
Scald	1	....	1	....	....	....	....	.....
Teeth, extraction of	3	2	5	....	....	....	....	.....
Tumefied earflap	4	....	4	....	....	....	....	.....
Tumors, unclassified	4	2	4	....	....	....	1	1
Wounds	48	10	55	1	....	2	....	.....
10. MISCELLANEOUS								
Autopsy	4	....	....	....	....	....	....	.....
Chill	1	....	1	....	....	....	....	.....
Debility	2	....	1	....	....	....	1	.....
Destroy	29	72	....	....	....	....	....	.....
Examination	8	....	....	....	....	....	....	.....
Goitre	2	....	2	....	....	....	....	.....
Observation	3	....	....	....	....	....	....	.....
Poisoning, phosphorus	1	1	1	....	....	1	....	.....
Poisoning, strychnine	1	....	....	....	....	1	....	.....
Rheumatism	12	....	8	3	1	....	....	.....
Rickets	9	1	8	1	....	....	1	.....
Undetermined	3	2	1	....	....	2	....	2
Vaccination, rabies	1	....	....	....	....	....	....	.....
	1,359	445	....	....	....	....	....	.....

Total cases	1,804
Counted twice	30
Net total	1,774

REPORT OF THE DIAGNOSTIC LABORATORY FOR THE YEAR ENDING JUNE 30, 1926

C. M. CARPENTER, C. J. PARSHALL, AND D. W. BAKER

Department of Comparative Pathology and Bacteriology

Although the general routine work of the diagnostic laboratory does not change from year to year, there are continually new problems for study being presented to us by the layman and veterinarian. Because of this fact, a certain amount of research must be carried out by this laboratory to aid in the diagnosis and prevention of such diseases as occur. These diseases must be studied when good material is available.

During the last year considerable time has been spent in research upon the relation of *Brucella abortus* infection in cattle to that in man. Eight cases of human infection with the Bang organism have been studied. Much work has been done in an effort to trace, if possible, the source of infection and compare the virulence of the human strains with that of those isolated from cattle and swine. Infectious abortion in cattle is very widespread and appears to be causing greater losses each year. More attention has been given this disease than heretofore because of its possible relation to disease in man and its economic importance to the owners of livestock. We feel that the agglutination test for this infection is of great value as a means of controlling the disease and therefore should be employed more generally. In the past many veterinarians considered the blood test to be worthless, or impractical, but as our knowledge concerning the disease has increased this test has been found to be extremely valuable. Laboratory examinations are in far greater demand than formerly and because many samples of blood are sent in unsatisfactory containers and received in poor condition it has been found necessary to study various types of bottles that are most satisfactory for the collection and shipment of blood to the laboratory. It has also been found more practical to supply, to those veterinarians who desire it, sterile rubber-stoppered bottles of the correct size and shape for the collection of blood samples. Because of lack of funds to employ sufficient help to make the blood tests and to purchase suitable sample bottles, the laboratory has been compelled to make a charge of 20 cents per sample to cover the cost of making the examinations.

During the last year (see Table I) a total of 6,104 examinations have been made, which is an increase of 3,688 over the number examined in the year 1924–1925. The greatest increase has been in the number of blood tests made for *Brucella abortus* infection, although there was an increase of approximately 500 examinations of other types.

There is appended to the report protocols and a review of the total number of specimens received for examination, the number of tissues examined for rabies, and the number of doses of tuberculin prepared and distributed by the laboratory, showing the general increase in the work of the Diagnostic Laboratory for the last twenty years.

The examinations for various diseases and the work done upon them during the last year are discussed below under the several sections.

RABIES

During the year 1925–1926 a total of eighty-one specimens were sent to the laboratory to be examined. Negri bodies were found present in twenty-six, or thirty-two per cent, of the total number. All of the positive cases occurred in dogs. This is the largest number of cases of rabies that has been examined and found positive at this laboratory since 1919–20 and only four less than the total of those occurring in the last five years. Rabies is at present on the increase in the State. For the most part the cases have occurred in the southeastern and western parts of the State. The infection seems to be spreading from these two areas, extending eastward from Buffalo and Niagara Falls and north and west from New York City.

The examinations were distributed among the various species of animals as follows: Dog 68; Cow 4; Guinea Pig 2; Sheep 1; Impossible 6.

GLANDERS

Eight samples of blood were sent to the laboratory to be examined for glanders, and were forwarded to the New York City Department of Health, by which the tests were made. Six were positive and two were negative to the test. The total number received last year was the same as was received during 1924–1925, but there was one less positive case.

INFECTIOUS ABORTION

During the year, 4783 blood tests were made for *Brucella abortus* infection (see Table 1). This is 3241 more tests than in the year 1924–25, or approximately three times as many. This would indicate that the test is becoming increasingly popular. Sixty samples of milk have been examined for the Bang organism. About fifty per cent of the samples have shown the presence of the infection. Studies have been made of the milk from cows which have been vaccinated with the living culture, as well as of milk from cows which have shown a negative and a positive blood test. The results of these studies have been published. They are as follows: The milk from cows which had been vaccinated three and four times with a living suspension of *Brucella abortus* showed the infection in thirty-eight per cent of cases. The milk from cows which had not been vaccinated but showed clinical

evidence of the disease (abortion or retained placenta), showed *Brucella abortus* in 66.6 per cent, while cows which had not aborted and had negative blood tests showed practically no infection.

Experiments upon the value of the agglutination test for infectious abortion in controlling the disease in badly infected herds are in progress. Reports of these findings will be made later. The positively reacting animals (those showing complete agglutination in a dilution of 1-60, or higher) are isolated from those animals which show no agglutination at the 1-60 dilution of serum. Tests are made upon the herds every sixty or ninety days. Records of the breeding history of the two groups are being kept so that a comparison of their efficiency can be made. In our work we have found that complete agglutination of a *Brucella abortus* antigen by serum diluted at 1-60 indicates infection. In general this criterion has been satisfactory and is above the range of the so-called "group agglutinins."

The genital tracts of several bulls have been examined for *Brucella abortus* and it has been found present in four. Last year it was reported that this organism had been recovered directly from the semen of a bull showing a positive blood test. This year a second bull has been examined which showed *Brucella abortus* in his semen. We have studied the breeding history of several bulls which show a positive blood test. Bulls infected with *Brucella abortus* are by no means rare. We have also studied twenty-six samples of semen for the presence of bacteria and other factors that might interfere with breeding efficiency.

Brief directions for the collection of blood samples in the agglutination test for *Brucella abortus* infection have been prepared and printed. Because many veterinarians were not familiar with this procedure it seemed necessary and expedient.

ANTHRAX, BLACKLEG, HEMORRHAGIC SEPTICAEMIA, ETC.

More examinations for hemorrhagic septicaemia have been made than during the previous year. Most of the cases have been observed in cattle, sheep and lambs. There have occurred two cases of blackleg and three cases of anthrax. Examinations have been made of several tissues and of milk for tuberculosis. A few cases of necrobacillosis have been diagnosed (see Table 1).

MISCELLANEOUS

The examinations of tissues for malignancy have increased as well as the number of samples of feces to be examined for parasitism. Several cases of Johne's disease have been observed. We have examined 218 samples of mastitis milk, the great majority showing infection with alpha streptococci. Quite a number of chemical examinations have been requested. These were turned over to one of the chemists in the University who is qualified to do this work.

Biologics Prepared and Distributed by the Diagnostic Laboratory

The laboratory has prepared and distributed 49,219 doses of tuberculin. This is a decrease of 3,227 doses (see Table II). The amount of subcutaneous tuberculin sent out each year is decreasing markedly. Less than half as much subcutaneous tuberculin was distributed this year as last. The majority of veterinarians prefer to use the concentrated tuberculins.

The amount of autogenous bacterins prepared, however, has shown a marked increase. The total amount distributed is 61,400 cc. which is approximately three times the amount prepared during the year 1924–25 (see Table II).

PRELIMINARY REPORT OF STUDIES OF BRUCELLA ABORTUS INFECTION IN MAN

As was stated in the introductory remarks of this report, much work has been done on *Brucella abortus* infection in man. The studies have been confined chiefly to the following four problems:

1. A bacteriological and serological study of the blood of patients suffering from undiagnosed fevers.

2. The source of *Brucella abortus* infection in man and a comparison of the biologic characters of the human strains with those recovered from cattle and from swine.

3. The mode of infection and the channel of entrance to the body.

4. A bacteriological study of fetuses and placentas from women in whom abortion has occurred and the etiological factor is unexplained.

During the last year samples of blood from patients suffering from undiagnosed fevers have been cultured. A serological examination for abortus agglutinins has also been carried out. Cultures for *Brucella abortus* have also been made from the blood of apparently normal individuals because it was feared that perhaps the Bang organism might be in the blood stream without producing any noticeable symptoms. Examinations for specific *Brucella abortus* agglutinins were also carried out upon these same samples.

Inasmuch as, in the eight cases studied, we have been unable to trace the source of infection to swine, goats, or cheese that may have been made from goat's milk, we have spent considerable time upon the examination of milk from herds of cattle where abortion infection is present and from which herds milk was consumed by the patients studied. In some herds strains of *Brucella abortus* extremely virulent for guinea pigs have been recovered. We have also injected pregnant heifers, free from *Brucella abortus* infection, with strains isolated from man and have always found them to localize in the udder following abortion. In one cow which was slaughtered six and one-half months following abortion due to injection of a human strain, *Brucella abortus* was still present in the milk. From this evidence it seems plausible that certain virulent strains of *Brucella abortus* may, through the medium of milk, infect man.

Mohler and Traum[1] reported finding *Brucella abortus* in the tonsil of a child. The senior author[2] observed that when calves were fed milk infected with *Brucella abortus*, the infection could be recovered from the lymph glands adjacent to, and draining, the mouth and pharynx. Because of these findings a bacteriological

[1] 28th Am. Rep., B. A. I., U. S. Dept. of Agr., Washington, D. C., 1911, p. 147.

[2] Cornell Vet., 1924, 15, p. 16.

examination for *Brucella abortus* is being made of diseased tonsils that have been removed. Cultures are made and guinea pigs are injected with tissue extracts from the tonsils.

Because of the finding of *Brucella abortus* agglutinins in the blood serum of women who had aborted, it was considered expedient to make a thorough bacteriological study of fetuses and placentas for *Brucella abortus* infection.

Material is being collected locally and from Bellevue Hospital, New York City, for these examinations. The results of some of these findings are appended to this report. The complete report will be made at a later date. Financial aid for a large part of this work has been granted by the Heckscher Fund for Research.

TABLE I

Examinations Made by Diagnostic Laboratory During the Year Ending June 30, 1926

MORBID CONDITIONS	Positive	Negative
Abortion, bovine (due to Brucella abortus)		
Agglutination tests—Total number, 4,783		
Bacteriological, fetuses	7	4
milk	29	31
placenta	7	1
uterine discharges	3	3
Bulls, genital tracts	4	....
Abortion, equine (due to beta hemolytic streptococcus)	2	....
Abscesses, bovine		
B. pyogenes	9	....
Streptococcus	....	....
B. coli	1	....
Pseudomonas pyocyaneus	2	....
Undetermined	8	....
Equine	....	....
Staphylococcus	3	....
Actinomycosis, bovine	5	6
Anthrax	3	22
Blackleg, bovine	2	2
Broncho-pneumonia		
Calf	6	....
Cow	5	....
Dog	1	....
Fox	6	....
Sheep	3	....
Swine	1	....
Chemical examinations	1	14
Fat necrosis	1	....
Feces examinations		
Cat	....	1
Dog	....	36
Belascaris	4	....
Hookworms	12	2
Toxascaris	1	....
Whipworms	4	....
Fox		
Belascaris	7	2
Hookworms	23	8
Whipworms	2	....
Horse		
Ascaris	2	5
Strongylus	11	....
Sheep		
Stomach worms	1	....
Bovine	....	1
Glanders		
Agglutination tests	6	2
Hemorrhagic septicemia		
Bovine	18	22
Ovine	8	5
Porcine	....	....
Rabbit	3	....
Goat	1	....

	Positive	Negative
Hog cholera	5	5
Johne's disease	4	10
Mange		
Bovine		
Sarcoptic	4	....
Psoroptic	9	....
Canine		
Follicular	1	....
Equine	....	8
Fox		
Otodectic	3	....
Mastitis, bovine		
Alpha hemolytic streptococcus	181	....
Beta hemolytic streptococcus	14	....
B. coli	3	....
Staphylococcus aureus	9	....
B. pyogenes	3	....
Mixed infection	8	....
Milk examinations	....	11
Milk counts, bacteriological	254	....
Necrobacillosis		
Bovine	3	1
Ovine	2	....
Parasitic diseases		
Ascariasis		
Canine	1	....
Fox	1	....
Lice		
Canine	1	....
Bovine	2	....
Swine	2	....
Equine	1	....
Lung worms		
Sheep	1	....
Swine	3	....
Nodular disease		
Ovine	9	....
Stomach worms		
Ovine	2	....
Tape worms		
Canine	2	....
Rabbit	8	....
Whip worms		
Sheep	1	....
Bots		
Rabbit	2	....
Plant poisoning	9	....
Posterior paralysis in lambs	17	....
Rabies		
Canine	26	42
Impossible	6	....
Bovine	....	4
Sheep	....	1
Guinea pig	....	2
Semen (bacteriological and microscopical examinations)	26	....
Sterility, bovine		
Chronic productive salpingitis	4	....
Ovaritis	7	....

Tuberculosis	Positive	Negative
Bovine		
Milk	1	19
Pus	....	4
Skin	2	....
Sputum	1	3
Tissue other than the skin	3	7
Tumors		
Carcinoma	9	....
Fibroma	3	....
Papilloma		
Bovine	2	....
Canine	1	....
Sarcoma	1	....
Miscellaneous examinations	160	....
Unable to make diagnosis	8	....
	1,037	284
Total		1,321
Serological examinations for infectious abortion		4,783
Grand Total		6,104

TABLE II

Biologics Prepared by the Diagnostic Laboratory and Sent Out During the Year Ending June 30, 1926

Tuberculin—subcutaneous (cc.)	12,208
intradermal (cc.)	4,131
ophthalmic (cc.)	4,492
Total (cc.)	20,831

(The above amounts of the various kinds of tuberculin represent 49,219 doses.)

Mallein (cc.)	10
Total	10
Autogenous vaccines and bacterins	
Hemorrhagic septicemia (cc.)	4,200
Mastitis bacterins	52,100
Miscellaneous bacterins	5,100
Total	61,400

TABLE III

Examinations for Rabies Made at the Diagnostic Laboratory of the New York State Veterinary College for Twenty Years, July 1st, 1906, to July 1st, 1926

Year	Total Examinations	Negri Bodies Present	Per Cent Showing Negri Bodies
1906–07	63	36	57.1
1907–08	315	188	59.6
1908–09	583	283	48.5
1909–10	329	143	43.4
1910–11	182	86	47.2
1911–12	180	85	47.2
1912–13	285	159	55.8
1913–14	490	252	51.4
1914–15	325	157	48.3
1915–16	108	48	44.4
1916–17	190	86	45.3
1917–18	207	91	44.0
1918–19	285	143	50.2
1919–20	122	48	39.4
1920–21	56	7	12.5
1921–22	39	6	15.4
1922–23	57	4	7.0
1923–24	72	5	6.9
1924–25	60	8	13.3
1925–26	81	26	32.1

TABLE IV

Amount of Tuberculin Prepared and Distributed by Diagnostic Laboratory for Twenty Years

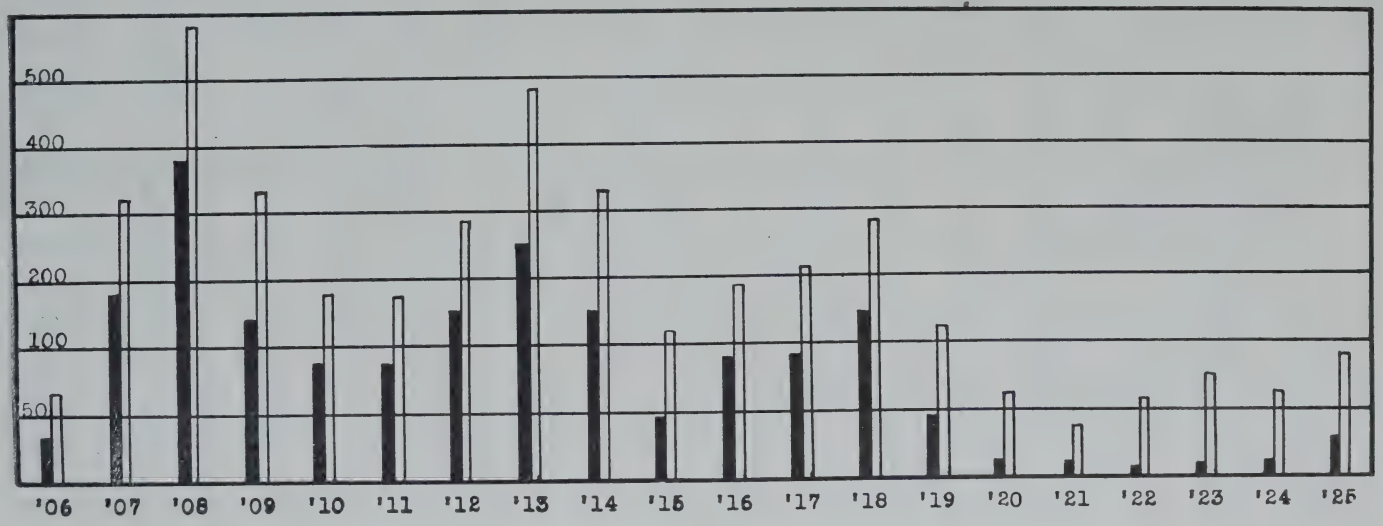
Year	Doses
1906–07	35,000
1907–08	35,889
1908–09	35,298
1909–10	43,275
1910–11	47,429
1911–12	55,681
1912–13	61,372
1913–14	61,065
1914–15	54,901
1915–16	55,602
1916–17	79,063
1917–18	72,007
1918–19	72,549
1919–20	77,006
1920–21	76,954
1921–22	68,381
1922–23	73,043
1923–24	73,432
1924–25	52,446
1925–26	49,219

TABLE V

Total Examinations Made by the Diagnostic Laboratory for the Last Twenty Years

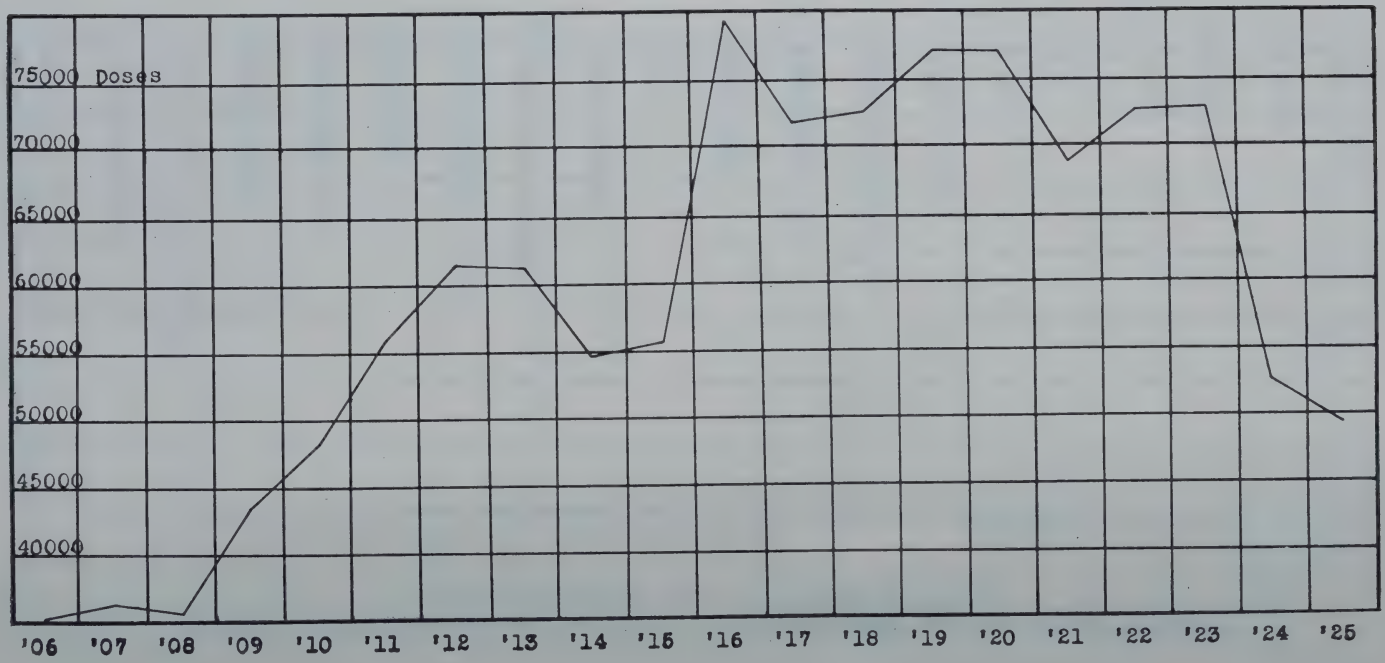
Year	Number of Examinations
1906–07	550
1907–08	656
1908–09	793
1909–10	924
1910–11	1002
1911–12	1038
1912–13	1658
1913–14	1770
1914–15	1089
1915–16	1039
1916–17	1364
1917–18	948
1918–19	1307
1919–20	968
1920–21	778
1921–22	989
1922–23	1742
1923–24	1586
1924–25	2416
1925–26	6104

GRAPHIC REPRESENTATION OF THE NUMBER OF EXAMINATIONS MADE FOR RABIES BY THE DIAGNOSTIC LABORATORY FOR THE LAST 20 YEARS

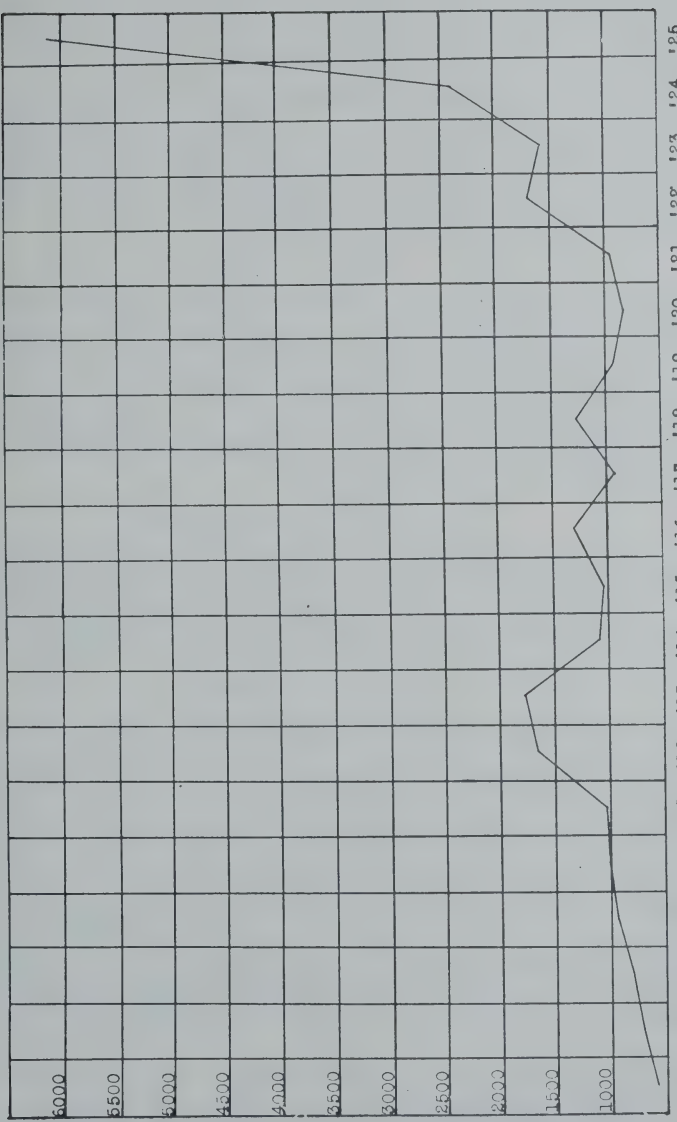


(Shaded areas represent the percentage of specimens showing the presence of negribodies.)

GRAPHIC REPRESENTATION OF THE NUMBER OF DOSES OF TUBERCULIN PREPARED AND DISTRIBUTED BY THE DIAGNOSTIC LABORATORY FOR THE LAST 20 YEARS



GRAPHIC REPRESENTATION OF THE NUMBER OF EXAMINATIONS MADE BY THE DIAGNOSTIC LABORATORY FOR THE LAST 20 YEARS



REPORT OF THE DIAGNOSTIC WORK ON POULTRY DISEASES AT ITHACA

JULY 1, 1925, TO JUNE 30, 1926

E. L. BRUNETT

Department of Pathology and Bacteriology

A total of 1,385 specimens were received and examined during the past year. These consisted of 1,325 chickens, 22 grouse, 11 geese, 9 turkeys, 8 ducks, 5 pheasants and 5 parrots. The total number of cases or accessions was 659.

FOWL PLAGUE

The last outbreak of this disease reported in this State occurred in June, 1925. One or two outbreaks were also reported about the same time from neighboring states. Since then the disease has not been found associated with poultry losses.

The control measures instituted after its recognition and the inability of the virus to survive very long outside the living tissues soon brought about its suppression.

BACILLARY WHITE DIARRHEA

Consideration is given to bacillary white diarrhea elsewhere in this report. The importance of *Bacterium pullorum* infection has not decreased. It still remains the most serious disease of baby chicks with which the poultrymen have to contend.

TUBERCULOSIS

The number of diagnoses of tuberculosis has remained essentially the same as last year. It must be remembered that the yearly diagnoses represent new foci of the disease. The fact that material received at the laboratory does not represent a large per cent of the actual trouble tends to make this particular disease seem less important than it really is.

In some flocks where tuberculosis was present, the intermingling of pigs and calves with the tuberculous hens was observed. The importance of the avian tubercle bacilli in certain types of tuberculosis of pigs and calves has been demonstrated by a number of workers. The relation of the disease in chickens to the tuberculosis problem in this State remains to be determined.

PARASITES

Parasitic troubles have been as prominent as in the past. There has been a sharp increase in the number of flocks infested with tapeworms, principally of the semi-microscopic variety. These infestations have been confined chiefly to the young stock, the losses appearing soon after they are placed in winter quarters. Losses have also occurred during other times of the year.

Numerous vermifuges have been experimented with, but their practical application remains yet to be demonstrated. More complete research work on the life cycles of poultry tapeworms is needed. The question of the intermediate host, or hosts, is one which when solved will enable poultrymen to control these pests. Until this information is forthcoming, continued losses from the ravages of these parasites must be expected. The losses each year which are caused by infestations with these worms will not allow their importance to be ignored.

During the year (Aug., 1925) one dead gosling was received from a nearby farm. The owner reported the loss of 100 goslings during the summer. Examination of the gosling revealed a heavy infestation of nematodes in the gizzard. Material received later confirmed the first diagnosis.

Specimens of the parasite were forwarded to Dr. M. C. Hall of the Bureau of Animal Industry who identified it as *Amidostomum anseris*, a species of the superfamily *Strongyloidea*. A description of this parasite by Eloise B. Cram, of the Zoological Division of the Bureau of Animal Industry, was published in the North American Veterinarian, Vol. 7, No. 1, January, 1926. The title of the article is "A Parasitic Nematode as the Cause of Losses among Domestic Geese."

This is the first published report of the presence of this parasite in this country. Its importance as a pest among geese cannot be overlooked. In the particular flock from which specimens were received, it constituted the only cause of the depletion of nearly the entire flock. It will be well to keep this nematode in mind when parasitic infestations of young geese are suspected.

Besides the routine work of making a survey of poultry diseases prevalent in this State, the following problems have been given special attention. The work has not been completed for publication in this report. Repetition of some of it is desirable and it is hoped that further work will furnish us with more knowledge about these particular subjects.

Cross-infection of adult male and female chickens infected with *Bacterium pullorum*.

Plumbism in chickens, acute and chronic types with some reference to metallic poisoning in general.

Bacterium pullorum infection with special reference to agglutination testing.

The appended table enables one to obtain some idea of the diseases present in our poultry flocks. The numbers to the right of the diseases refer to the number of flocks in which the particular disease was diagnosed. In practically all cases the disease listed was the cause of the unusual flock mortality at the time the material was examined.

DIAGNOSES

DISEASE	CHICKENS		Turkeys	Grouse	Geese	Ducks	Pheasants	Parrots
	Young	Adult						
Specific bacterial diseases:								
Bacillary white diarrhea—								
Bact. pullorum infection	73	11	....	....	....	....	....	.....
Bact. sanguinarium infection	2	....	....	....	....	....	....	.....
Fowl cholera	....	6	....	....	....	....	....	.....
Psittacosis	....	....	....	....	....	....	....	1
Tuberculosis	....	16	1	....	....	....	....	.....
General bacterial diseases:								
Abscess	....	1	....	....	....	....	....	.....
Air-sac infection	....	1	....	....	....	1	....	.....
Bumble foot	....	1	....	....	....	....	....	.....
Edema of wattles	....	3	....	....	....	....	....	.....
Pericarditis	....	4	....	....	....	....	....	.....
Peritonitis	....	9	....	....	....	....	....	.....
Pneumonia	....	....	....	....	....	....	1	.....
Subcutaneous infection	....	1	....	....	....	....	....	.....
General diseases:								
Ascites	....	1	....	....	....	....	....	.....
Dilatation, crop	....	1	....	....	....	....	....	.....
Dilatation, heart	....	1	....	....	....	....	....	.....
Enteritis	....	3	1	....	....	....	....	1
Hemorrhage, comb	....	1	....	....	....	....	....	.....
Hemorrhage, internal	....	9	....	1	....	....	....	.....
Hemorrhage, ovarian	....	1	....	....	....	....	....	.....
Impaction, crop	....	1	....	....	....	....	....	.....
Impaction, gizzard	....	3	....	....	....	....	....	.....
Impaction, intestine	....	2	....	....	....	....	....	.....
Impaction, oviduct	....	7	....	....	....	....	....	.....
Internal layer	....	11	....	....	....	....	....	.....
Intestinal stricture	....	1	....	....	....	....	....	.....
Intestinal twist	....	3	....	....	....	....	....	.....
Leukemia and pseudo-leukemia	....	12	....	....	....	....	....	.....
Obesity	....	1	....	....	....	....	....	.....
Periostitis	....	2	....	....	....	....	....	.....
Poisoning, lead	....	1	....	....	....	....	....	.....
Poisoning, metallic, undetermined	2	....	....	....	1	....	....	.....
Prolapse, cloaca	....	6	....	....	....	....	....	.....
Rupture, esophagus	....	2	....	....	....	....	....	.....
Rupture, ova	....	12	....	....	....	....	....	.....
Tongue necrosis	....	1	....	....	....	....	....	.....
Traumatism	....	2	....	2	....	....	....	.....
Tumors	....	35	....	....	1	....	1	.....
Ulcers, crop	....	....	....	1	....	....	....	.....
Ulcers, gizzard	....	4	....	....	....	....	....	.....
Diseases of the head:								
Avian diphtheria, bronchitis tracheitis	....	37	....	....	....	....	....	.....
Chicken pox	....	4	....	....	....	....	....	.....
Roup	....	5	....	....	....	....	....	.....
Nutritional diseases:								
Rachitis	....	1	....	....	....	....	....	.....
Visceral gout	....	14	....	....	....	....	....	.....
With esophageal pustules	....	4	....	....	....	....	....	.....
With nephritis alone	3	6	....	....	....	....	....	.....
Parasites:								
Flukes, intestinal	....	....	....	....	....	1	....	.....
Lice	....	5	....	....	....	....	....	.....
Mites:								
Cytodites nudus	....	2	....	....	....	....	....	.....
Sarcoptes mutans	....	1	....	....	....	....	....	.....
Roundworms:								
Amidostomum anseris	....	....	....	....	1	....	....	.....
Capillaria	1	27	....	....	....	....	....	.....
Gizzard worm, unidentified	....	2	....	....	....	....	....	.....
Heterakis perspicillum	1	31	....	....	....	....	....	.....
Heterakis vesicularis	3	11	....	....	....	....	....	.....
Tapeworms	2	111	1	....	....	1	....	.....

DIAGNOSES

DISEASE	Chickens		Turkeys	Grouse	Geese	Ducks	Pheasants	Parrots
	Young	Adult						
Protozoan diseases:								
Coccidiosis	43	5	....	....	....	....	....	.....
Entero-hepatitis	7	....	5	....	....	....	....	.....
Fungus diseases:								
Aspergillosis	1	....	....	2	....	....	....	.....
Favus	....	1	....	....	....	....	....	.....
Miscellaneous:								
Decomposed	13	13	1	....	....	....	....	.....
Negative examination	32	20	....	2	2	2	1	.....
Normal	....	6	....	....	....	....	....	.....
Unclassified	7	15	....	1	....	1	1	.....
	190	497	9	9	5	6	4	2
Counted twice	5	58	....	....	....	....	....	.....
	185	439	9	9	5	6	4	2

REPORT OF THE POULTRY DISEASE DIAGNOSTIC LABORATORY AT FARMINGDALE, LONG ISLAND

July 1, 1925, to *June* 30, 1926

J. M. HENDRICKSON
Department of Pathology and Bacteriology

The intensity and extent of the poultry industry on Long Island are enormous and are being commercialized to the highest degree. The industry is growing rapidly and steadily. As in all industries of this kind, it is hard to get exact figures, but in 1920 it was estimated that there were 20 to 25 chicken farms carrying 2,000 or more layers. Today there are over 250 commercial chicken farms with an average of 1,000 birds per farm. There are several farms that carry 10,000 layers throughout the year. One of these large farms utilizes 150 acres of land for its poultry. The above figures do not include the baby chick phase of the industry, where the figures run from a few hundred to 100,000 chicks marketed by a single poultry farm during the season. The annual value of the chickens and eggs raised on Long Island may be conservatively estimated at $3,000,000.

The largest duck industry in the world is centered on Long Island. There are over 100 duck ranches, the largest of which marketed 180,000 ducks last year. It is estimated that approximately 3,500,000 ducks are raised on the Island each year, with a value of over $5,000,000.

As disease and disease problems multiply with the increase in numbers and concentration of any living organisms within a restricted area, so in the chicken and duck industry on Long Island the problems of disease, its prevention, control and treatment have become more and more acute from year to year as the industry has increased in volume. The occurrence of fowl plague here during the fall of 1924 and spring of 1925 and the large losses incurred each year from other maladies have placed many of the poultrymen in a morbid state of despondency.

The poultry disease diagnostic laboratory was established at the State Institute of Applied Agriculture, Farmingdale, Long Island, last July by the New York State Veterinary College at Ithaca, under a cooperative agreement between the two institutions. The object of the laboratory is to find out what the diseases and disease problems are; to find means of prevention, control and treatment for the diseases found to exist; and to assist the poultrymen in every way possible to overcome disease in their flocks and place them on a more solid foundation in this respect. There is no doubt that the poultry industry on Long Island can be increased many fold if disease can be controlled.

During the year there has been received at the laboratory a total of 427 accessions, or lots of birds, for diagnosis. The total number of birds autopsied was 1,036, which includes 867 chickens, 157 ducks, 1 pigeon, 1 gosling, 3 pheasants and 7 turkeys. There were also 1,004 agglutination tests made for the detection of *S. pullora* carriers in adult birds. The large amount of correspondence, telephone calls, conferences with poultrymen and veterinarians, visits to poultry farms, and other work which necessarily come with work of this type, are not recorded in this report.

The experimental and research work done in connection with specific diseases has been rather limited, due to the fact that the amount of material received for diagnosis has been so great, and the matter of starting and equipping a laboratory and also becoming acquainted with the disease problems that confront the poultrymen on the Island, have left very little time for work of this character. However, some investigational work has been done, particularly on roup, chicken pox, infectious bronchitis, fowl typhoid and bacillary white diarrhea. The nature and results of this work will be taken up in the discussion of the various diseases.

BACILLARY WHITE DIARRHEA

The losses from this disease are tremendous, and it is one of the biggest scourges with which the poultryman has to contend. Forty outbreaks were recorded at the laboratory during the year, most of which were associated with high mortality. The great development of the baby chick trade and the just demand of the purchasing poultryman that the chicks must be free from white diarrhea have placed a heavy bond on the hatcheries and poultrymen who make a business of hatching and selling day-old chicks on the market. Very little work has been done in this State in regard to the application of the agglutination test to eliminate carriers of this disease in the adult birds used for breeding purposes. The facilities open to the poultryman who desires to have work of this character done are entirely inadequate and he is unable to compete in the baby chick market with the surrounding states which have been carrying out a careful program for the eradication of bacillary white diarrhea for a number of years. Something will have to be done to overcome this situation and prevent the great economic loss to the poultrymen of New York State. A movement is being started and plans are being formulated whereby, in our present light of knowledge, we think that bacillary white diarrhea may be eliminated and a breeding flock maintained which will be entirely free from the disease. This work will be developed during the coming year.

During the year five flocks of chickens, where bacillary white diarrhea had been the cause of losses in chicks to a greater or lesser degree in previous years, were tested by means of the agglutination test and all reactors removed and the place thoroughly cleaned and disinfected. Records, as complete as possible, as to

fertility, hatchability and mortality in previous years, were obtained on the flocks and the owner was requested to keep similar records on the results following the elimination of the reactors and cleaning up of the premises. Of the five flocks tested, only one owner has sent in a partial report to date on the results this season. This man had a loss of about 40 per cent in his young chicks two years ago, 25 to 30 per cent a year ago, and this year only about 2 per cent, of which the greater number of deaths have been due to toe pecking, drowning, etc. When tested, 34.4 per cent of the birds in this flock gave a positive agglutination reaction. The work on these flocks will be continued during the coming year and some additional flocks will be included in an attempt to determine the efficiency and reliability of the test.

PARASITES

The losses from intestinal parasites are very great, especially where large numbers of birds are kept over a period of years in close confinement. Growing birds are especially susceptible to this malady. It is interesting to note that, besides the cases listed in the table in which these various parasites were diagnosed as being the specific cause of the trouble, the large intestinal roundworm (H. perspicillium) was found to be present as a complicating factor in 47 cases; the cecal worm (H. vesicularis) in 31 cases; capillaria in 11 cases, and tapeworms in 22 cases. If we add these figures to those in the diagnosis table, we find that in the 427 accessions received at the laboratory during the year, the large roundworm was present in 74 cases, or 17.3 per cent; the cecal worm in 38 cases, or 8.9 per cent; capillaria in 24 cases, or 5.6 per cent, and tapeworms in 55 cases, or 13 per cent.

ROUP AND CHICKEN POX

These two conditions are very prevalent on Long Island and are the causes of great economic losses to the poultrymen. The loss usually is not due to the high mortality in the affected flock, but rather to the loss in egg production as a result of the disease. It was found that a large number of the poultrymen were vaccinating their birds each fall with various so-called avian mixed bacterins as a preventative and also using these bacterins as a therapeutic measure when outbreaks occurred.

On October 5 three birds were brought to the laboratory which showed characteristic lesions of chicken pox on the comb and wattles. There were also some diphtheritic lesions in the mouth and pharynx, and evidence of roup. The flock from which these birds came numbered about 400 and was kept in a long house which was partitioned off into four compartments, containing about 100 birds each. The birds in compartment No. 4 were affected the most, about 25 per cent showing lesions. In the other three compartments only a few of the birds were affected, and these not badly so. In another house nearby were about 125 pullets, a few

of which showed poxlike lesions, and close to these was a house containing about 150 to 200 old hens that did not show any lesions. The cockerels out in the range houses did not show any lesions.

In October 6 the scabs and crusts from the birds in compartment No. 4 of the long house, where the outbreak was the most marked, were removed and used for the preparation of an autogenous bacterin. At the same time the scabs were removed, the parts were cauterized with a stick of caustic potash and where roup was present the eyes and nostrils were washed out with a boric acid solution and a few drops of 15 per cent argyrol solution applied.

Fifty of the birds in compartment No. 2 were then vaccinated with ½ cc. of the autogenous bacterin and four days later were given a second injection of ½ cc. The other 50 birds in the compartment were left for checks. At the same time 50 birds in each of compartments Nos. 1, 3 and 4, were vaccinated with a commercial avian mixed bacterin which was procured on the market and was the same as is being used by many poultrymen. Fifty birds in each compartment were left unvaccinated as checks. Pen No. 1 was given three doses of ½ cc. each, three days apart. Pen No. 3 was given a ½ cc. injection and three days later an injection of 1 cc. Pen No. 4 received a single injection of 1 cc.

A careful check was kept on all pens. The results in brief were as follows: There was a general improvement in all pens for a few days following the vaccination, which was noted in vaccinated and non-vaccinated alike. Fourteen days after the vaccination there was a sudden severe outbreak of chicken pox and roup in all pens and there was no material difference in the number and severity of the cases in the vaccinated group and those that were left as checks. By November 16th the disease had run its course and cleared up with no difference between vaccinated and non-vaccinated birds noted at any time. On February 8, there was another general outbreak of colds, roup and bronchitis in all four pens with vaccinated and non-vaccinated affected in a like manner.

In this experiment the use of an autogenous bacterin or the commercial avian mixed bacterin which was used, was of no value as an immunizing or therapeutic agent for roup or chicken pox.

INFECTIOUS BRONCHITIS

Infectious bronchitis was the cause of large losses during the fall and winter months. The disease is characterized by its sudden onset, gasping for breath with marked rattling and wheezing sounds, and ending in death or recovery in a few days. The more chronic cases have the symptoms of roup with formation of heavy mucous and caseous exudates and is indistinguishable from it. The chief lesions on post mortem are found in the trachea and bronchi and vary from a severe hemorrhagic inflammation of the mucous linings to a complete plugging of the trachea and bronchi with clotted blood and mucus. No specific etiological agent could be demonstrated and all attempts to transmit the disease artificially, from the material received at the laboratory, failed.

In flocks that were left unattended, the disease spread rapidly and with heavy mortality. It was found that if the birds were taken as soon as the first symptoms appeared and placed in warmer quarters, under strict hygienic and sanitary conditions, with proper nutrition, the termination was usually rapid and favorable. Fifteen grains of equal parts of ammonium chloride and ammonium carbonate given once or twice daily as an expectorant, to assist in the elimination of the exudate, were found very beneficial. Equal parts of the chloride and carbonate of ammonia were also used with beneficial results in a wet mash at the rate of one ounce to 25 or 30 birds as a flock treatment, though the individual treatment with capsules is more satisfactory. These drugs have also been used in other respiratory troubles with beneficial results when used properly.

FOWL TYPHOID

One outbreak of fowl typhoid was encountered in a flock of approximately 10,000 birds. The mortality had ranged from 80 to 150 birds per day for about four days when the birds were brought to the laboratory. The place was in a very insanitary condition and the owner was advised to thoroughly clean and disinfect all buildings and equipment and remove all affected birds as soon as noticed. Permission was obtained from the owner to vaccinate 50 per cent of the birds with an autogenous bacterin prepared in the laboratory from the freshly isolated typhoid organism. However, by the time the bacterin had been prepared, the mortality had stopped, apparently as a result of the hygienic and sanitary measures and the removal of affected birds, in all but one house, containing about 300 birds, where losses were still occurring, though not as frequently as they had been. One hundred fifty of these birds were vaccinated with 1 cc. of the autogenous bacterin, but the disease rapidly subsided and there was no difference noted in vaccinated or non-vaccinated. The application of strict hygienic and sanitary measures was apparently all that was necessary to check this outbreak.

DISEASES IN DUCKS

The losses in young ducklings during the past season have been very great. One duck man lost 30,000 ducklings during the month of February and did not rear a duck that was marketable during this period. This same condition was true on many other duck ranches and losses occurred by the hundreds daily. The losses were mostly in young ducklings from a few days to one month in age. Very little trouble was encountered in old ducks and breeders.

The cause of the losses was apparently due to a number of factors. *Bact. anatum*, the etiological agent of so-called white diarrhea in ducklings, was isolated from the internal organs of dead ducklings quite consistently. Improper nutrition, faulty brooding and housing, poor ventilation, and unhygienic and insanitary conditions, were found to play important roles in losses on the various ranches.

DIAGNOSES

DISEASES	Chickens		Ducks		Pheasants	Goslings	Pigeons	Turkeys
	Young	Adult	Young	Adult				
Specific bacterial diseases:								
Bacillary white diarrhea	40	....	....	....	....	....	....	....
Bact. anatum infection	....	....	12	....	....	....	....	....
Bact. pullorum infection	....	3	....	....	....	....	....	....
Fowl typhoid	....	1	....	....	....	....	....	....
Tuberculosis	....	2	....	....	....	....	....	....
General bacterial diseases:								
Abscess	....	2	....	....	....	....	....	....
Bumble foot	....	2	....	....	....	....	....	....
Edema of wattles	....	1	....	....	....	....	....	....
Pericarditis	....	2	....	....	....	....	....	....
Peritonitis	....	17	....	....	....	....	....	....
Pneumonia	2	2	....	....	....	....	....	....
General diseases:								
Ascites	....	1	....	....	....	....	....	....
Bile duct occlusion	....	5	....	....	....	....	....	....
Broken egg	....	5	....	....	....	....	....	....
Cannibalism	....	4	....	....	....	....	....	....
Egg bound	....	5	....	....	....	....	....	....
Enteritis, necrotic	....	2	....	....	....	....	....	....
Frozen comb and wattles	....	2	....	....	....	....	....	....
Hemorrhage, external	....	1	....	....	....	....	....	....
Hemorrhage, internal	....	5	....	....	....	....	....	....
Hemorrhage, liver	....	9	....	....	....	....	....	....
Hemorrhage, ovarian	....	3	....	....	....	....	....	....
Impaction of crop	2	3	....	....	....	....	....	....
Impaction of oviduct	....	12	....	1	....	....	....	....
Internal layer	....	13	....	....	....	....	....	....
Leukemia or pseudo-leukemia	....	4	....	....	....	....	....	....
Obesity	....	1	....	....	....	....	....	....
Ovarian trouble	....	2	....	....	....	....	....	....
Poisoning	....	1	....	1	....	....	....	....
Prolapse of oviduct	....	5	....	....	....	....	....	....
Tumor	....	23	....	....	....	....	....	....
Typhlitis, necrotic	....	1	....	....	....	....	....	....
Diseases of head and respiratory system:								
Avian diphtheria	....	9	....	....	....	....	....	....
Chicken pox	2	7	....	....	....	....	....	....
Infectious bronchitis	....	24	....	....	....	....	....	....
Roup	3	33	....	....	....	....	....	....
Roup with canker	....	2	....	....	....	....	....	....
Nutritional diseases:								
Nutritional disease with esophageal pustules	1	3	....	....	....	....	....	....
Nutritional disease with nephritis alone	8	8	....	....	....	....	....	....
Nutritional disease with rachitis	....	....	5	....	....	1	....	....
Articular gout	....	1	....	....	....	....	....	....
Visceral gout	....	17	....	....	....	....	....	....
Parasites:								
Lice	2	1	....	....	....	....	....	....
Mites:								
Dermanysus gallinae (red mite)	1	1	....	....	....	....	....	....
Laminosioptes cysticola (connective tissue mites)	....	1	....	....	....	....	....	....
Sarcoptes levis (depluming mite)	....	1	....	....	....	....	....	....
Roundworms:								
Capillaria (small intestinal)	10	3	....	....	....	....	....	....
H. perspicillum (large intestinal)	14	13	....	....	....	....	....	....
H. vesicularis (cecal)	4	3	....	....	....	....	....	....
Tapeworms	23	10	....	....	1	....	1	....
Protozoan diseases:								
Coccidiosis	31	....	....	....	1	....	....	1
Entero-hepatitis	6	....	....	....	....	....	....	1

DIAGNOSES

DISEASES	CHICKENS		DUCKS		Pheasants	Goslings	Pigeons	Turkeys
	Young	Adult	Young	Adult				
Miscellaneous:								
Killed by dog or fox	3	....	....	....	....	....	....	.....
Mechanical injury	....	3	....	....	....	....	....	.....
Decomposed	1	3	....	....	....	....	....	.....
Normal	....	1	....	....	....	....	....	.....
Unclassified	11	2	....	1	....	....	....	.....
	164	285	17	3	2	1	1	2
Counted twice	26	20	....	....	1	....	....	1
	138	265	17	3	1	1	1	1

THE INTERPRETATION OF CLINICAL SYMPTOMS*

By D. H. UDALL

Department of Medicine, Obstetrics and Ambulatory Clinic

When Dean Moore suggested that I discuss some phase of physical diagnosis, on account of his belief that the present tendency is towards too great dependence upon the laboratory, I presume he was thinking along the line that recently suggested the following from the pen of a noted physician: "At one time the physician did occupy a dominant position, but during the last fifty years the laboratory workers, and the surgeons, have made such progress in their special departments that they usurp the physician's position. To a great extent the physician is himself to blame. He has allowed himself to be dazzled by the inventions of his colleagues, and has accepted their methods and ideas, not recognizing the limited scope of all such methods, while his own vastly more important field has been neglected."

A disease presents three chief problems: the origin, the cause, and the prognosis. The last is the most important, and by some is regarded as the chief aim of all medical effort.

A disease is recognized by its symptoms. In every case some action or condition of the subject shows that functions are disturbed, or tissues damaged. This may be recognized by a special test, such as tuberculin, or by observation. One first seeks evidence on the *environment,* the *history,* the physical symptoms or *signs,* and the *cause.* Knowledge thus obtained always has been and probably always will be our chief dependence in diagnosis and treatment. In most cases it constitutes our sole dependence. All other evidence must harmonize in its interpretation with such knowledge. The manifestations of disease in the living are the only continuous expressions of that disease; to overlook or ignore them is to invite error.

Environment

Regard for environment is, in one way or another, of use in the diagnosis, the prognosis, and the treatment of every sick individual. Symptoms of primary indigestion in pastured cows are nearly always signs of traumatic gastritis. In this locality, acute affections of the digestive tract, such as indigestion, gastroenteritis, and displacements, are most common, as well as most fatal in the last quarters of the year, while impactions predominate in the first quarter. Diseases of the young, such as white scours, tend to increase in morbidity and mortality from month to month, during the winter in any given herd. Malignant head catarrh in cattle occurs chiefly in certain restricted localities, in the spring. In wet seasons parasitic diseases are more destructive

* Reprinted from The Cornell Veterinarian, April, 1926. Vol. XVI, p. 137.

to calves and sheep. In tuberculous herds with a continuous high percentage of reactors, the older cows that have been in the herd for years, merit special attention. Affections of the reproductive organs, of the udder, or of the newborn, have a varying significance according to the farm where they occur. Azoturia and pneumonia in the horse often take a mild course in farm horses, while they are more fatal in cities.

HISTORY

History as related to disease may be divided into three groups: family history, past and immediate history of the individual. Scant credit has been given to the transmission of disease from one generation to another in animals. Observations recorded by Dr. W. L. Williams and others upon genital disease in certain families have indicated its occurrence. It is not unusual to find in a herd that a certain family has a bad reputation as breeders, and that very little increase has occurred during four or five generations. On investigation of one such group I learned that about fifty per cent of the conceptions through five generations had terminated abnormally. Such evidence is an indictment of the family. Theobald Smith[1] in a recent report on intra-uterine infection of the lungs with *B. abortus* states that in newborn calves: "pneumonic lesions due to *Bacillus abortus* may maintain the virus in the body as long as a month, according to these studies, and may perhaps account for the occasional discovery of infected unbred heifers. In such cases the bacillus may be carried to the udder and there it may survive indefinitely."

Until recently the histories of individuals have played a minor part in supplying information on disease. Most chronic affections have been of such a nature that once symptoms became manifest, they remained so, *e. g.* tuberculosis in cattle, and glanders in equines.

Development of the purebred cow has increased the demand for continuous breeding from the same animal. In this field a knowledge of the history of the individual is of great value as an index to health. Such a history includes information on the number of services necessary for conception, and the number of progeny. While an examination of the fetal membranes may reveal both the past and the future. Regard for this information is of inestimable value to one who contemplates the addition of a new individual to a healthy herd, or the purchase of animals for the purpose of establishing a breeding herd.

Recurrent digestive troubles suggest chronic peritoneal adhesions. In cows this frequently results from a traumatic gastritis. Unthriftiness in cows since a previous parturition suggests traumatic gastritis, or a chronic metritis. Recurrent fever in equines suggests infectious anemia and glanders. A history of dummy-like symptoms in a horse should lead one to suspect hydrocephalus,

[1] J. Exp. Med., 1925, xli, 639.

or tumors of the brain. In general, the history assists in the recognition of chronic disease, and the establishment of this condition alone is a long step towards an accurate diagnosis and prognosis. Nosebleed in the horse suggests chronic disease of the heart; in the cow, pulmonary abscess. This is evidence that may be brought out in the history and in no other place.

The *immediate history* includes all that has transpired since the onset of the present attack until the time of examination. To some it doubtless seems elementary to mention so obvious a fact, yet it is a common fault of both veterinarians and physicians to ignore many significant incidents associated with the onset of a given disease. There seems to be a natural tendency to regard disease in segments rather than as a continuous process. Knowledge of the onset brings one close to the cause. In handling affections of the young, knowledge that a given animal was weak at birth is the most significant fact of all; it justifies the conclusion that it is the offspring of a diseased dam. Temperature reading of an apparently normal animal before shipment, or in the presence of an enzoötic, is a useful application of this principle. The recognition of disease in its first stages may reveal the only information that can be utilized in its control. Information covering the twenty-four hours directly preceding an attack of indigestion or pneumonia may reveal as much as the clinical symptoms and the autopsy combined. Knowledge of the presence of cottonseed meal in special grain mixtures for calves may make it possible to explain an unthrifty long-haired condition that cannot be explained by a physical examination alone.

General or Constitutional Symptoms

Since the sensations experienced by a sick animal cannot be imparted to us, disease is expressed by objective symptoms or signs.

Attitude

The attitude may serve as a pathognomonic symptom, one that surely establishes the diagnosis of the disease. For example, the characteristic attitude of milk fever in conjunction with a normal or subnormal temperature is generally accepted as proof of the malady. Yet this conclusion is subject to error, for a septic metritis in cows may cause an identical attitude and temperature. Differentiation rests on an examination of the uterus, since the reddish pus that characterizes this type of metritis may not find its way to the vagina. When such a patient is still pregnant a diagnosis may be difficult.

A study of the meaning of attitudes is worth while. Certain attitudes in equines suffering from colic usually mean a dilated stomach. These are: a dog-sitting position; sternal recumbency with the front feet advanced; and a standing position with the head hanging nearly to the floor and the front legs slightly spread. On autopsies of horses that have assumed a dog-sitting posture, rupture or torsion of the colon may be found, but in such cases

gastric dilatation may have been present also. A stretched position is characteristic of impaction of the colon in equines and sheep, and is seen in intestinal intussusception in cows.

Lateral recumbency, a condition where the animal is down and unable to rise, may be due to various groups of causes. In the first group one would include exhaustion from indigestion, from being cast, and from fractures of the limbs. In another group one would include peripheral paralysis as observed in botulism or forage poisoning, and azoturia. In another, loss of consciousness as observed in encephalitis, lead poisoning, septicemia and milk fever. When an animal is down it is important to learn whether it is conscious, whether it takes notice of the surroundings, and whether it can chew and swallow if food is placed in the mouth.

An example of the significance of recumbency is observed in botulism. In 1900 Professor Leonard Pearson[2] reported a study of general paralysis in horses that had eaten mouldy silage. He reproduced the disease in experimental horses by feeding. Until this time such paralytic symptoms had served for the diagnosis of "cerebrospinal meningitis," but Professor Pearson made note of the fact that on autopsy no lesions were found in the brain or spinal cord; it was not a disease of the central nervous system, and he suggested the name "forage poisoning." Subsequent studies have revealed botulinus toxin, an agent that causes symptoms and autopsy changes like those described by Pearson. The disease produced by it is termed botulism; it has definitely recognized characteristics. Except as applied to botulism the term forage poisoning has no definite meaning, though other toxins may yet be discovered in the food or water.

What relation has this to the attitude? In well-developed botulism or forage poisoning, paralysis is general. In encephalitis paralysis is general. But the victim of botulism is conscious, while the victim of encephalits is unconscious. This elementary sign alone serves to distinguish one from the other. Yet twelve years after Pearson's report, the largest outbreak of epizoötic encephalitis recorded in this country received the official diagnosis of "Forage Poisoning." Thousands of horses manifesting deranged consciousness from the onset to the end were pronounced victims of this intoxication.

In most affections the attitude is filled with meaning to those who are seeking for signs, and while a sign may not furnish conclusive evidence, every sign is a measure of proof that demands interpretation.

Changes in the Color of the Skin and Mucosae

The significance of the common changes in the mucosa are too familiar to merit extensive discussion. These are the *icterus* of duodenal catarrh, of rapidly progressive anemia as seen in swamp

[2] J. Comp. Med. and Vet. Archives, 1900, xxi, 654.—A Preliminary Report upon Forage Poisoning of Horses (so-called Cerebro-Spinal Meningitis.)

fever, and of certain of the infectious diseases such as influenza, and contagious equine pneumonia. In the infections it is sometimes interpreted as a sign of intestinal catarrh, when in reality it is due to rapid dissolution of the blood—a grave condition. Icterus combined with deranged consciousness is very suggestive of disease of the liver. This is seen in cows suffering from acute fatty degeneration of the liver.[3] Icterus and convulsions in pigs usually mean ascaris invasion of the bile ducts. Icterus and convulsions in the horse have been reported from invasion of the bile duct with a larva of *Estrus equi.* The presence of hemorrhagic spots on the conjunctival sclera of young calves bespeaks an impending diarrhea. Similar changes were observed in impending encephalitis in horses in Kansas in 1912. The diffusely red conjunctival mucosa of horses suffering severely from intoxication of indigestion, displacement of the intestines, or septicemia is well known. It does not appear that cows affected in a similar manner present the same circulatory change in the visible mucosae.

Fever

The dictionary definition of fever states that it is "an abnormally high body temperature," and the height of the temperature is supposed to represent the height of the fever. In cattle a fever is common with no rise of temperature, so that one must depend on other signs, such as chills, cool horns, teats, and extremities, increased pulse rate, the appetite, the sensorium, and a dry muzzle.

When a rise of temperature is present one first asks, what are the groups of causes that may produce it? What diseases does it eliminate? and how may it have been modified by the length of time that the animal has been sick? Is it a symptom that characterized this disease from the onset, or is it a later development like the temperature of a gastro-enteritis or lead poisoning?

A high temperature at the onset of a disease has a more definite meaning than one that may be found on a first call after the animal has been sick for some days. In general, with the bovine as a subject, *high temperature at the onset* may be due to the following groups of causes:

1. Septicemic diseases with no definite localization; general infections, some of which may localize. Anthrax always. Blackleg always. Hemorrhagic septicemia nearly always. Necrobacillosis of the peritoneum, pleura, and uterus nearly always carries a high temperature. Septicemia due to certain other microörganisms that operate occasionally, do not spread by direct or indirect contact, and about which little is known as a cause of septicemia in cows—colon bacillus, *Bacillus pyocyaneus.*

2. Local infections, some of which may become general. Wound infection, infrequent. Metritis sometimes causes a high temperature, which always indicates a severe infection; but there are so

[3] Cornell Vet., 1926, xvi, 64.

many exceptions that a normal registration on the thermometer has, by itself, no significance — the course may be rapidly fatal, or mild. Malignant head catarrh, always. Mastitis often causes a high temperature, but there are many exceptions. Traumatic gastritis often causes a low degree of fever; it is rarely high, and it may be absent. Primary pneumonia: fever always present at the onset. Secondary pneumonia: fever often absent.

3. Primary indigestion: fever frequent, either high or low without regard to severity.

4. Disturbance of heat regulation: sunstroke.

5. Poisoning. In much of the literature on poisoning in animals the condition with respect to fever seems to have been overlooked. It is sometimes written that differentiation between metallic poisoning and a general infection can be made by the absence of fever in poisoning. One of the most frequent forms of metallic poisoning, lead, may cause a temperature of 105° or more within twenty-four hours. In a recent Federal Bulletin, stock poisoning by twenty different plants, including ferns, is discussed, but in a casual examination I have found no reference to temperature in the discussion on symptoms. In another bulletin on "The Poisonous Properties of Whorled Milkweed," the temperature is reported at 108.5°, and higher. In another bulletin that mentions death from feeding of a plant experimentally, no mention is made of the temperature or any other symptom. In an extensive bulletin on the study of goitre in animals I have not found any reference to the temperature. In this connection it is interesting to note that Cabot, in discussing the causes of fever, mentions toxemia without infection and gives exophthalmic goitre as an example. In literature on sweet clover poisoning, Broerman of Ohio states that the temperature readings were negative, and Fitch of Minnesota that the temperature is normal or subnormal. In a bulletin on loco poisoning I have not found mention of the temperature. Forage poisoning is stated by Mohler, in U. S. Dept. Agriculture Bul. 65, to have a temperature of 103° to 107°. All other writers that I have consulted (Meyer, Hart, Hayes) emphasize the fact that the temperature in botulism is either normal or subnormal. This apparent conflict is due to the fact that Bulletin 65 is based on the description of a disease that was not forage poisoning.

Infections and intoxications with normal or subnormal temperature at the onset:

1. General infections: purpura hemorrhagica in equines, often.

2. Local infections that may lead to septicemia, toxemia, or pyemia: wound infection (traumatic gastritis); metritis; mastitis; actinomycosis; gastro-enteritis (numerous exceptions); secondary pneumonia.

3. Toxins: *B. botulinus* toxin.

4. Poisoning: sweet clover.

Summing Up

After the general examination is complete, one can usually decide whether the subject is affected primarily with a general disease—one affecting many parts or all parts of the body, such as anthrax, blackleg, or hemorrhagic septicemia. If during the course of a fatal disease no symptoms arise that point to the seat in some system, this may be found on autopsy; if the autopsy is largely negative, one's conclusion must still be based on the symptoms; if a laboratory examination is negative, one's conclusion must still be based on the symptoms. In diseases of a general nature, conclusions based on negative findings after the patient is dead are of meagre value. To illustrate methods of summing up evidence, three cases are submitted.

Case No. 1.

A 4-year-old cow was taken suddenly sick on the afternoon of November 8. The next morning she was leaning forward in the stanchion, feet tucked under the body, the head lowered and carried to the right. During the examination she fell to the knees. Pawing. Profuse salivation. Ptosis of the right upper lid. Pulse 96. T. 101.8°. Examination of respiratory and digestive tract negative. November 10, paralyzed.

Autopsy Nov. 11. Abomasum and small intestines diffusely hemorrhagic over entire mucosa. Mesenteric lymph glands hemorrhagic. Lungs congested and emphysematous. Pericardium and myocardium presented diffuse hemorrhage. The meninges of the left side of the cerebellum were highly congested. A few small hemorrhages were found on the floor of the 4th ventricle, the brain stem, and the lining of the infundibulum. *Bacteriological examination:* A rabbit injected with lung extract died of hemorrhagic septicemia. Cultures from the spinal fluid were negative.

Diagnosis. Is this primarily a general infection, a gastro-enteritis, or an encephalitis? Clinically it was an encephalitis as revealed by the paralysis and ptosis. Yet the cow could swallow and readily took a quart of mineral oil. This suggests that the brain lesions, meningitis and encephalitis, were not in themselves of sufficient severity to be so rapidly fatal. The most marked lesions were in the digestive tract, and preference is given to this system as the primary location of the trouble, because the digestive mucosa is a frequent primary seat of disease, while the central nervous system frequently is involved in a secondary process. It is not usual to find severe gastro-enteritis secondary to acute inflammations of the brain. As an initial and dominant clinical manifestation, brain symptoms have been observed in pulmonary abscess, traumatic gastritis, and metritis in cows. The temperature of 101.8° has no special significance, though it is opposed to the view of a primary general infection. It is not rare for both the cow and the sheep to die suddenly without showing definite clinical signs, and on autopsy to reveal enteritis. The presence of *Bacillus bovisepticus* in the lung can hardly be accepted as evidence that the cow died of hemorrhagic septicemia, though this was considered as a provisional clinical diagnosis.

Case No. 2.

A 6-year-old Guernsey calved May 28. Placenta removed April 1. The cow seemed normal until the morning of April 3, when she showed anorexia, dullness, a tucked-up abdomen, and thin watery feces. Pulse 80. Breathing 45-50. T. 105.6°. Rales over the right lung. The uterus contained a reddish fetid fluid, and sloughed cotyledons. April 8 she died of septic metritis and pneumonia. *Autopsy:* The lungs showed lesions typical of hemorrhagic septicemia, and *B. bovisepticus* was found in a practically pure culture. The uterus contained streptococci and *B. necrophorus.* In this, as in the preceding case, the hemorrhagic septicemia organism had nothing to do with the disease except as a secondary factor. All of our cases of metritis that have been infected with the necrosis bacillus have carried a high temperature, and have ended fatally.

Case No. 3.

A group of heifers aged about 8 months developed a high temperature, persistent diarrhea, and edematous swellings of the limbs. Several died in the course of a few days. All of the clinical evidence in the case pointed towards a general infection. On autopsy the *pleura* was red and a slight fibrinous exudate was present in two or three intercostal spaces. Spleen normal. *Heart* covered with hemorrhages. *Lungs* showed marked congestion in the ventral and anterior lobes. *Liver* normal. *Gall Bladder:* bile very thick and full of flakes; mucous membrane ulcerated and hemorrhagic. *Kidneys* swollen. *Bladder* normal. *Intestines* full of yellow mucus. *Omasum* contained small hemorrhages.

The autopsy suggested a general infection. No single organ presented lesions sufficient to cause death, and no individual system was the chief seat of the disease. The clinicians thought that the disease was of the nature of a general infection, possibly hemorrhagic septicemia, and treated it accordingly. Bacteriological examination failed to reveal *Bacillus bovisepticus* and this negative evidence was presented as proof that hemorrhagic septicemia did not exist.

Here is a case where the clinician is justified in seriously considering the possibility that the subjects were victims of hemorrhagic septicemia regardless of the fact that the bacteriological examination was negative. In the other two, where it was positive, an opposite conclusion seems more logical. The first question to answer is, were these animals affected with an infectious disease, or some form of poisoning? The clinical evidence alone is sufficient to exclude the probability of poisoning from any substance that exists in this part of the country. If infectious, it was an enzoötic in young animals with no visible evidence of the channel of infection. It could not be explained by localization of disease in any one organ. Blackleg was excluded. Anthrax was excluded. There remained one other well-known general infection — hemorrhagic septicemia. The symptoms were not unlike those that have

been described in standard texts for this disease. It is true that other microörganisms may affect a group of animals in a similar manner, but the reports of such instances are infrequent in veterinary literature. Up to the present time there has been no general linking of the bacteriological laboratory with routine veterinary practice, and it is certain that infectious diseases of which we now have no knowledge will eventually be described. A clinical diagnosis is an opinion based on the evidence at hand. In this group, positive evidence of the exclusion of *B. bovisepticus* does not exist, for it is not infrequent that this particular bacillus escapes detection when present in animals that it has destroyed.

CONVULSIONS IN DOGS

H. J. MILKS

Department of Materia Medica and Small Animal Clinic

During the last few years and more particularly during the past year convulsions or fits in one form or another have caused us considerable worry and from the reports of other localities they have caused considerable concern throughout the state and nation.

A convulsion may be defined as an involuntary contraction of a muscle or group of muscles. They are much more common in young than adult animals and in small than large animals.

The same grade of intoxication, or irritation, in the young will cause a more acute motor reaction, with disturbance of consciousness, than in the adult. Thus a convulsion may replace a chill in the infectious fevers of young animals and especially young small animals.

Saltmann in experimental work on animals has thrown some light upon the subject. Some of his more important findings are:

1. The irritability of the motor neurons of the new born is negative and that of the sensory low; both varieties develop beyond a normal irritability and reach an electrical hyperirritability above that seen in the adult.
2. The cerebral motor area is undeveloped at birth and does not react to electrical stimuli.
3. The reflex inhibitory centers and paths are lacking at birth.
4. The development of the inhibitory function does not keep pace with that of the motor function and the peripheral irritability.

While there have been objections to some of Saltmann's conclusions it is generally believed that the inhibitory function is absent at birth and develops slowly.

There is also in older animals a certain volitional motor control that is undeveloped in young ones and consequently the untrained nervous system of the young is more easily thrown off balance than in the adult. There is no doubt that there is less of this volitional control in animals than man and certainly less in young than aged animals. Thus in the young we have a lacking of the automatic inhibitory mechanism plus any volitional control. In other words, there is a lack of balance between the excitant and inhibitory forces.

It is not easy to explain the why or whys of a convulsion or to understand what is known as a "reflex convulsion." A consideration of the spasms of strychnine with which you are all familiar may be of some help in understanding them. We commonly say that strychnine causes convulsions; and yet strychnine, in itself, does not cause them, but needs some stimulus, however small, which is always available. Strychnine convulsions are reflex in

nature, that is, they are a reaction to some stimulus. In an ordinary reflex action only certain groups of muscles are involved because the afferent impulses under normal conditions only pass through certain motor cells. Under the influence of strychnine these afferent impulses not only pass through the usual motor cells, but spread through other paths to other motor cells and reach and affect cells ordinarily beyond their influence. Under normal conditions a reflex is a purposeful reaction to some stimulus. If we approach a normal animal and stir him up by clapping the hands, he will react by performing some act to see what is wanted or what the excitement is about. If he has had a moderate dose of strychnine, he will jump or, as we say, go on his toes over the same stimulus, and if the dose is large enough he will go into a convulsion. We explain the action of strychnine by believing that it so facilitates the passage of afferent impulses through the cord that an ordinary reflex act is exaggerated into a convulsion.

Predisposing causes or constitutional condition. These include any condition affecting the nutrition of the brain, since cerebral irritability is greatly aggravated by such disorders. Holt states that any disturbance of nutrition such as exhaustion, anemia, malnutrition, debility or any acute disease, especially of the digestive tract, may predispose to convulsions.

Rickets is believed to be an important factor. On account of its retarding the growth of the nervous system it renders this system more susceptible to peripheral irritation. Holt claims that convulsions may be the first symptom of rickets in children, and that when convulsions occur without evident cause, rickets should be looked for. We have not checked this up to any extent in dogs, but have often observed convulsions in rickety pigs.

Diseases of the internal glands, notably the thymus and thyroid, have been incriminated by some practitioners. I have made no observations along these lines and am unable to discuss this point.

The excitant causes may be divided into:

1. *Direct irritation of the brain cortex.* This group includes all convulsions due to true cerebral disease, tumor, hemorrhage, inflammation, etc.

2. *Peripheral irritation or reflex causes.* Some difference of opinion exists as to the importance of this group, some holding that the group is important in itself and others that the different members are important only as they lower vitality and cause a depression of nerve tone sufficient to bring the nervous system to a convulsive reaction, — in other words, that this group increases the susceptibility and, where this is great, the cause may be trivial.

There are arguments for and against each member of this group, but the difference of opinion about them is of minor importance from a therapeutic view since it makes no difference whether it is indigestion itself or its products acting upon the centers that cause the fits, or whether it is the irritation from the different worms or

the toxins liberated by them. In any event, in one case we must treat the cause of the indigestion and in the other remove the parasites.

The conditions most often set down as the causes of reflex convulsions are:

Dentition	Severe pain	Excitement
Indigestion	Phymosis	Over exertion
Intestinal parasites	Parasitic otorrhea	Unknown causes

Of these, to my way of thinking, dentition is not a common cause. This is a normal process and usually occurs without much inconvenience to the animal. I can understand, however, how at times it might cause trouble.

Parasitic otorrhea is a common cause of convulsions in cats, and occasionally in dogs. The French find this so common that they call the disease "contagious epilepsy." In describing the symptoms of parasitic canker of the ear, Cadiot says, "In a number of individual convulsions, signs of auricular giddiness, and epileptiform crisis are observed at different intervals. At the first these accidental reflexes appear only occasionally as when the sick dog is excited by running or during the hunt. The dogs appear sly, uneasy, anxious, rush straight ahead; groan, or cry and fall prey to convulsions. These crises occur at intervals of several weeks and finally more and more frequently. Some of these sick dogs become timorous or apathetic, and some morose and aggressive."

Toxic conditions. These include asphyxia, infectious diseases and, particularly, distemper, about which I have nothing to offer.

Emotional. Fright, or disturbance, may be a factor but we have not seen cases in which these factors could be incriminated except in case of distemper or other conditions which were, so to speak, set on a convulsive hair trigger and ready to explode with the slightest disturbance.

Unknown cause. This group includes every case upon which we can find nothing. Many of them may be true epilepsies and others may be due to irritations which we are unable to find.

Prognosis. A convulsion or fit must always be regarded as a serious condition. While few animals die of the attack, if treated promptly, relieving the fit or treating the symptoms is not sufficient since more convulsions are likely to follow unless the cause is discovered and removed. We believe that many unexplained sudden deaths among dogs are due to convulsions but, of course, cannot support this theory. We have had many instances of these sudden deaths. One of our clients lost three or four full grown setters at intervals of a few days or weeks. They were all well as usual at night and were found dead in the morning. Poisoning was out of the question. An autopsy of the last two showed nothing but a heavy infestation of hookworms and treatment of the rest of the dogs for this trouble solved the riddle. Question: If these dogs did not die of a fit, what killed them?

Treatment: This must include emergency treatment for the convulsion and the removal of its cause, if this can be determined. This, of course, goes back to a careful examination and diagnosis. Frequently nothing can be discovered and no active treatment, aside from relieving the convulsion, is indicated. Of course some sedative is often prescribed to tide these animals over their crisis and frequently that is all that can be done in convulsions of unknown origin. In those in which the causative factor is believed to have been discovered, treatment should be directed to the removal of this irritant.

So much for convulsions in general. I have gone over this subject because we feel that there is very little difference between a convulsion proper and a running fit, or frights disease, other than some possible difference in the centers involved or in the response to the irritant. At any rate treatment must be very similar. You are all more or less familiar with the symptoms shown by dogs with frights disease. They start suddenly, look about wildly, then run straight away as fast as they can go, barking and crying as though terribly frightened, as they are. They act like a person in delirum, see things which are not there and try to get away from them and hide. Sometimes they run to their masters for protection. After one attack they appear worried, timid, anxious, and but little is required to give them another start. In kennels of several animals it is not unusual to have the attack in one animal start several or all the rest going and then there is something doing for everybody concerned. Consciousness is not lost. Instead of running into obstacles they are more apt to run home or to crawl under things and hide. Sometimes these running spells are succeeded by true convulsions.

Following our line of thought that this condition was similar to convulsions or epileptiform attacks, we tried to find a cause for them, but have not every time been successful. We have, with considerable success, connected the disease with roundworms, hookworms, tapeworms, indigestion, over exertion or fright, parasitic otorrhea and distemper.

Many times we have not been able to demonstrate the cause.

Our belief in a connection between the above conditions and this disease, is based on the ground that in many cases the attacks ceased when these conditions were cleared up.

A few case reports illustrating this belief follow:

Roundworms: Case I. A 7 months old, spayed German shepherd bitch, in excellent condition. She had a history of having had these attacks for two or three days, in fact, spoiled the owner's vacation. Physical examination was negative. Feces examination showed a heavy infestation of round worms. *Treatment:* Oil of chenopodium and castor oil. Results: No more trouble.

Case II. A three or four months old King Charles Spaniel. She had had several such attacks. At time of examination was very timid. As in case I, physical examination did not help. Feces ex-

amination gave a heavy ascaris infestation. *Treatment:* She was given a dose of barbital sodium to keep her quiet and 5 minims of oil of chenopodium in castor oil. Results: Recovered.

Indigestion: Case I. Spayed Airedale three or four years old. This animal had one of the typical running fits at home and, of course, scared the owner and his wife. Owner was about to shoot her on one of her trips through the house, but thought better of it and caught her instead. He was advised over the telephone to give her two ounces of castor oil. There was no further trouble.

Case II. Boston terrier one year old. She had a history of having had several of these spells on the 7th of November. We saw her on the 8th. She was very nervous and easily excited. Physical examination and feces examination negative. We administered one ounce castor oil and kept her under barbital sodium for a day or two, after which she had no further trouble.

Tapeworms: Case I. A six months old hound which had thrown several running fits under no particular or unusual conditions. Physical and feces examination were negative except for presence of many tapeworm segments in feces. After treating for tapeworms there were no further signs of fits.

Case II. Another hound handled and treated in the same way with similar results.

Hookworms: Instances of recovery from this affection following the diagnosis and treatment for hookworms, have been so common that it seems almost unnecessary to cite the cases. To be specific, one typical case was a six months old hound that had had several running fits. Physical examination negative. He had no further trouble after the hookworms were removed. In many instances several dogs in the same kennel have cleared up nicely after diagnosis and treatment for these parasites.

Parasitic Otorrhea: Case I. A beagle bitch and three or four puppies would go into these convulsions at the sight of food. Physical examination was negative except for parasitic otorrhea in each animal. This was treated and the disease disappeared promptly.

Case II. A pack of pointers was reported in a very bad way. One of the animals was brought to us in a very weakened condition, having had convulsions almost continuously during the previous day. We could not find anything wrong with him except a bad case of parasitic ear canker. He was treated for this but did not do well. He cleared up for a time and was sent home where he had a relapse and the disease finally terminated in true convulsions. On autopsy he showed the so-called "nervous lesions of distemper." He did not show any symptoms of distemper while in the clinic. In the other eight or nine dogs belonging in this kennel, the local veterinarian succeeded in finding the ear parasites in all but one, and this was his first attempt at diagnosing the condition. The rest of the animals, except one, cleared up after treatment for otorrhea. None of them were reported to have had distemper.

Distemper: The usual case we see is as follows: The dog, usually a young one, is apparently all right, suddenly becomes frightened, runs, barks and cries. In some instances they go into dark corners or under things to hide. Upon the first examination nothing abnormal is found except shyness, and perhaps a slight elevation of temperature. Within a few days he has more of these attacks and in 4 or 5 days, or less, he will show typical signs of distemper. A few will recover, but we ordinarily consider it a bad omen. In other instances they follow an apparently light case of distemper and here the prognosis is even more dubious since there is a strong tendency to develop into true convulsions and the animal either dies, or is in such a condition that he is put out of his misery.

I will not worry you by citing cases of distemper as you are all more or less familiar with this phase of the disease.

Excitement and Over Exertion: Case I. An adult setter in good condition but soft, having had little exercise. She had these running spells each time she was taken after pheasants. Physical and special examinations were negative. We advised more regular exercise but do not know the results.

Case II. A spayed Boston terrier about one year old. Ran away from home in the residential district and became confused in the busy part of the city. She had one of these attacks in a department store and was treated by another practitioner who gave her a hypodermic of morphine. After two or three days she had another attack but has since been all right.

I do not want to give the impression that it is always easy or possible to determine the cause or to handle every case, as we find many in which we are unable to discover anything wrong or to give much relief. Neither am I certain that the cases discussed under the last head should not belong in the unknown group, although it appears to us that many dogs which have little opportunity to hunt, except on a few isolated days, are so afflicted. Neither do I desire to have you believe that we know all that is necessary about this troublesome condition. What I have endeavored to point out is the connection of the disease, or symptom, with several other well-known diseases.

A COMPARISON OF THE BLOOD OF A NORMAL AND TWO CASTRATED BILLY GOATS

PIERRE A. FISH AND CHARLES E. HAYDEN
Department of Veterinary Physiology, Cornell University

Two of the goats were twins born Feb. 5, 1923. One of these was used as a control; the other was castrated at the age of 74 days. A third kid, born April 1, 1923, was castrated at the age of 83 days and added to the experiment.

Examinations of the blood were not begun until about a year after the castrations. There were eighteen chemical examinations covering the period of a year, with an interval of about three weeks after each examination. There were twelve blood counts covering a period of eight months, also at three-week intervals. In all cases the blood was taken from the jugular vein.

The data obtained are given with some reserve and are offered only tentatively as a report of progress. One apparent effect of castration is weakened resistance. About three months before the blood examinations were begun, both castrates, then about one year of age, developed a skin affection, which for lack of more precise knowledge is referred to as a "dermatosis." This condition persisted while the examinations were made. Also at about the time the blood work was begun, there was reason to believe that the castrates were afflicted temporarily with stomach worms — *Haemonchus contortus*. The entire was apparently exempt from both of these afflictions. This point of difference should be kept in mind in the comparison of results. During some of the colder months the animals were brought to the laboratory to be bled. It is not impossible, in this connection, that the moderate exercise involved, with possibly some nervous excitement, might cause some rise in the sugar content of the blood.

The methods of Folin and Wu were followed, and the sugar readings were always taken promptly. Some differences in the uric acid readings were due to the fact that after the work was begun a purer preparation of sodium tungstate was obtained and this reduced the quantity found. The blood counts were made from the blood which had been oxalated for the chemical examinations. In the warm season the goats were allowed to run in a paddock, but during the cold months they were kept under shelter.

The following tables show the results obtained:

Chemical examination (18)
(Milligrams per 100 cc. of blood)

	Sugar	Total non protein nitrogen	Urea (17)	Uric acid	Creatinin	Chlorides
Entire	62.44	35.78	18.47	0.325	1.27	492.2
Castrates (average of two)	59.78	32.83	15.66	0.348	1.07	524.4

Blood counts (12)

	Hemoglobin	Erythrocytes	Leucocytes	Lymphocytes -%	Mononuclears -%	Polynuclears -%	Eosinophiles -%	Basophiles -%
Entire	90	15,225,000	6,158	45.9	6.08	43.5	3.7	0.7
Castrates (average of two)	84.1	13,911,500	12,141	44.1	3.38	41.3	9.8	1.25

The following table from Welsch (1) shows the normal blood count average from five male and five female goats.

	Erythrocytes	Leucocytes	Lymphocytes -%	Mononuclears -%	Polynuclears -%	Eosinophiles -%	Basophiles -%
5 male goats	13,390,000	9,800	55.	4.	40.	1.	1.
5 female goats	14,490,000	8,080	42.	6.	50.	2.	1.

The following data on sheep are taken from Sustchowa. (2)

3 male sheep	13,060,000	uncastrated, 1–1½ years.
5 male sheep	9,420,000	castrated 1–2 years.
5 female sheep	9,780,000	normal.

In the chemical examinations, it will be noted that the margin is usually in favor of the entire, and the differences, for the most part, are within the normal range of variation. The chlorides, however, were almost invariably higher in the castrates.

The chlorides of the blood are believed to be associated with the production of HCl in the gastric secretion and chlorides are also eliminated through the urine. It may be inferred that a higher chloride content in the blood of the castrates may signify a decreased amount of HCl, in the gastric juice, thus influencing the digestive process in the stomach, or a reduced urinary elimination of chlorides. Actual tests for either of these possibilities were not made.

The influence of the carbon dioxide tension of the blood upon the chlorides in the plasma may also be a factor. According to T. Brailsford Robertson (3): "When whole blood is treated with CO_2, the chlorides in plasma decrease, and the bicarbonates increase, while on decreasing the carbon dioxide tension, the reverse phenomena occur. Evidently hydrochloric acid, free or combined flows into the cells under high CO_2 tension and leaves them again

when the CO_2 tension falls. Simultaneously bicarbonates flow out of the cells under high CO_2 tension and enter them again when the CO_2 tension falls.''

In the blood counts, the number of erythrocytes was greater in the entire than in the castrates. Sustchowa noted a similar difference in entire and castrated sheep, with a more pronounced difference in the sheep. The number of leucocytes in the castrates was double that of the entire. The percentage of hemoglobin and mononuclear leucocytes was higher in the entire, but there was a higher percentage of eosinophiles and basophiles in the castrates, possibly associated with the dermatosis.

The general statement is made by G. Müller (4) that males have more hemoglobin than females and castrated animals more than entires. In the blood of the goats less hemoglobin was found in the castrates than in the entire.

Lipschütz (5), quoting Ocaranza, states that a diminution in the number of red corpuscles occurs after castration, which agrees with the result of the present experiment.

After assembling the preceding data, an article by Sustchowa on the influence of age, sex and castration on the number of the red corpuscles and hemoglobin content in cattle, swine and sheep, and one by Welsch on the blood of swine, sheep and goats, were available. In these articles, and others, the indication seems to be that the data relate to single examinations of the blood taken from a greater or less number of animals and the average for the group taken as the result. References to the work of other investigators are made in the above articles, and the work of some of these has been used in comparison with our own results. Although our own work was limited to a small group of three goats, the continued observations over a long period of time, it is believed, have some advantages over single examinations of larger groups.

In general the effects of castration upon the blood of animals of other species, as recorded by other workers, agree with what was found in the goats.

Entire and Castrated Animals

Authority	Animals	Erythrocytes	Leucocytes	Hemoglobin
Storch (6)	2 Stallions	8,205,000	10,478	.....
	7 Geldings	7,595,000	11,020	.....
	10 Bulls	6,503,000	7,841	.....
	10 Oxen	6,683,000	9,367	.....
Sustchowa	9 Bulls	8,101,000	.....	80
	5 Oxen	6,910,000	.....	68
Storch	5 Rams	11,183,000	9,720	.....
	8 Wethers	9,839,000	9,642	.....
Sustchowa	3 Rams	13,060,000	.....	56
	5 Wethers	9,419,000	.....	58
Welsch	3 Rams	11,770,000	6,440	.....
	2 Wethers	11,360,000	8,470	.....
Sustchowa	5 Pigs (entires) females	9,976,000	.....	65
	3 Pigs (castrates) females	8,307,000	.....	67
	4 Pigs (castrates) males	9,400,000	.....	62
	NORMAL GOATS			
Storch	5 Billy goats	15,300,000	11,520	.....
Welsch	10 Billy goats	13,390,000	9,800	.....

Burnett (7), in a summary of examinations of the blood of normal goats by different investigators, shows a range of 9,000,000 to 19,000,000 erythrocytes and 8,000 to 12,000 leucocytes. His list also quotes 30,000 leucocytes by Hayem, but this number seems to us quite abnormal.

Storch found more erythrocytes and fewer leucocytes in the stallion than gelding. He found no marked difference in the erythrocytes of bulls and oxen (castrates) but noted more leucocytes in the oxen.

Sustchowa found more erythrocytes in the bull but did not investigate the leucocytes. He also found more hemoglobin in bulls. Storch, Sustchowa and Welsch, all found more erythrocytes in rams than in wethers. Storch found the number of leucocytes about the same in each but Welsch found more leucocytes in the wethers. Sustchowa found a slightly higher hemoglobin content in wethers. Sustchowa found a higher number of erythrocytes in normal entire female pigs than in castrated female pigs, and this held true also for castrated male pigs, when compared with the normal females.

Including our own work, a summary of the results indicates that after castration there is a reduced number of erythrocytes and an increased number of leucocytes. The hemoglobin content is higher in bulls and billy goats (entires), but in sheep and pigs it is slightly higher in the castrated animals. Whether an increased number of leucocytes follows as a direct result of castration, or whether the condition is associated with a lower number of erythrocytes, has not been investigated.

More data are being accumulated which we hope to include with this preliminary contribution, in a later paper.

Seasonal Changes. Because the blood counts were not continued through the whole year and because of the dermatosis in the castrates, a detailed report seems inadvisable until more complete observations can be carried out on another group of goats.

On the incomplete data we have and under the conditions mentioned, it may be of interest to note that the sugar content appeared to be higher, and the number of leucocytes slightly higher, during the cold season; while the number of erythrocytes and the percentage of lymphocytes were higher during the fall and spring seasons.

General Considerations. The blood is the main transportation system of the animal body. It traffics in various kinds of merchandise. It must travel on exact schedule time, for delay would wreak disaster to the tissues it serves. Food, fuel and oxygen must be delivered exactly when needed if the cells are to breathe and maintain their integrity. The freight of waste matter must be shipped at once from the cells, for its accumulation is destructive to them. There is no halt for loading or unloading; the exchange must be made while the blood is in motion.

It is from this basic fluid of the body that the various glands draw the raw material which they elaborate into their finished

products — the secretions. In the case of the endocrine or ductless glands, those which produce internal secretions or hormones, the blood not only furnishes the raw material but receives the finished product and transports it to the parts requiring it. For the welfare of the body, the internal secretions are quite as important as any of the other secretions. It is known that the internal secretion of the adrenal glands is essential to life. The disease or removal of these glands is fatal. The blood is no longer able to deliver a product, in this case very infinitesimal in amount, to the tissues which are absolutely dependent upon it.

The glands of the endocrine system vary in importance. The disease or removal of the pituitary, thyroid or parathyroid glands produce profound effects. The decreased amount or absence of the hormone insulin from the pancreas and its relation to diabetes, has attracted much recent attention. The hormone from the interstitial cells, associated with the sex glands, has been shown to have an influence upon temperament and bodily condition. The blood deprived of any of its hormones is an abnormal blood, and its relation to the tissues on which the hormone acts is correspondingly changed.

A soil deficient in quantity or in the absence of certain constituents may nevertheless produce crops, but these will likewise be deficient in quantity or quality. In a similar way the various tissue crops or activities of the body will suffer if deprived by disease or operative interference of any of the constituents which nature intended them to have.

Our acknowledgments are due Dr. Gibbons of the Department of Pathology for making the blood counts.

REFERENCES

1. WELSCH, W., Das Blut der Haustiere mit neueren Methoden Untersucht. V. Untersuchungen des Schweine, Schaf, und Ziegenblutes. Pflügers Arch. f. d. ges. Physiol. Vol. 198, 37–55, 1923.
2. SUSTCHOWA, N., Untersuchungen über den Einfluss des Alters, Geschlechts und der Kastration auf die Zahl der roten Blutkörperchen und den Hämoglobingehalt bei Rindern, Schweinen und Schafen. Arch. f. Anat. und Physiol. Physiol. Abt. 1910, 97–112.
3. ROBERTSON, T. BRAILSFORD, Principles of Bio-Chemistry. 2nd edition, 1924. p. 310.
4. SMITH, F., Manual of Veterinary Physiology. 5th edition, 1921. p. 14.
5. LIPSCHÜTZ, A., The Internal Secretion of the Sex Glands. p. 33.
6. STORCH, Ueber den Blutkörperchengehalt des Blutes laudwirtschaftlichen Haussäugetiere. Inaug. Dissertation. Karlsruhe. 1901.
7. BURNETT, S. H., The Clinical Pathology of the Blood of Domesticated Animals. 1908. p. 45.
8. MYERS, V. C., Practical Chemical Analysis of Blood. 2nd edition. 1924.

THE AGGLUTINATION TEST AS AN AID IN HANDLING BANG ABORTION DISEASE

R. R. BIRCH AND H. L. GILMAN
Veterinary Experiment Station

SUMMARY OF ESSENTIALS

The conception of Bang abortion disease which is represented in the following summary has been obtained by making agglutination tests and securing coinciding breeding records of all individuals in many New York dairy herds; by keeping under observation an experimental herd of approximately fifty animals for a period of four years, during which time monthly (and in some cases weekly or daily) agglutination tests have been made on each individual, and the results checked with breeding records and the appearance of *Bact. abortum* in uterine exudate, milk, and fetus; by special experiments dealing in particular with methods of transmission, development of immunity, and the relation of the agglutinin titre of the blood to various phases of the infection; by examining material secured from several breeding herds in the vicinity of Ithaca through cooperation with the ambulatory clinic; and, finally, by constant reference to the work of others, and acceptance of that which is well confirmed by reliable workers.

BANG ABORTION DISEASE

1. There is a specific infectious disease caused by *Bact. abortum*, which organism attacks the chorion, and is responsible for a high percentage of abortions among cattle in this and other states.

2. The disease is transmissible from cow to cow, through association of "spreaders" with susceptible animals.

3. In the infected cow the organisms usually localize in the lymph glands, udder and spleen; and, if the animal is pregnant, they localize in the chorion and invade the fetus. In bulls, which are much less frequently infected, the organisms are known to localize in the seminal vesicles, testicles, and epididymes.

4. From the infected cow the organisms escape in the chorion, and in the uterine discharges which precede and follow expulsion of the fetus, or calf, and they are often contained in the milk continuously, or intermittently, for months or years.

5. The organisms do not as a rule remain longer than five weeks in the uterine discharges following expulsion of the fetus.

6. At the first parturition following infection, as determined by serological tests, the fetal membranes (and fetus in the event of abortion) in almost all cases, contain *Bact. abortum*.

7. In subsequent pregnancies of the infected cow the organisms are localized in the fetal membranes much less frequently (20 per cent of our cases).

8. The organisms are not always killed during passage through the digestive tract.

9. Susceptible pregnant cows readily contract the infection through association with spreaders. Artificial infection terminating in abortion is readily brought about by feeding cultures of *Bact. abortum* to pregnant cows, and natural infection probably takes place through the same channel.

10. It is known that an occasional infected bull discharges *Bact. abortum* with the seminal fluid. Presumably cows may be infected by copulating with a bull thus infected. How bulls become infected is not well known.

11. The incidence of retained placenta, metritis, and sterility is much higher in a *Bact. abortum* infected herd than it is in a clean herd handled in the same manner, when the females of both herds are bred to the same bull.

12. A considerable number of individuals resist the infection altogether, or at least in a degree which will permit a perfect breeding record.

13. As a result of uterine infection brought about either by natural or artificial means, most individuals develop immunity, resistance, or tolerance in a degree that permits a satisfactory breeding record, even when they are exposed during subsequent pregnancies in a manner that will infect susceptible pregnant cows and cause them to abort.

14. The pregnant cow not previously infected is highly susceptible, her chorion being an ideal medium for growth of the Bang organism. There is good evidence that non-pregnant cows are less susceptible, but this point needs further study.

15. Heifer calves, reared in badly infected herds and taking milk from infected cows, rarely contract permanent infection. If these calves are removed from the infected herd before they reach sexual maturity, and protected subsequently, a very high percentage of them will breed satisfactorily, and show no evidence of *Bact. abortum* infection.

16. Other things being equal, a herd free of *Bact. abortum* infection is by far the most efficient breeding unit; but a herd consisting of previously-infected females or those carrying infections of long standing, provided such females will conceive, is a fairly satisfactory unit. While a herd is in the transition period between these two states, that is, while it is going through the infection toward the immunity, or tolerance, which lies beyond, the losses from abortion and associated ills are enormous.

17. In all breeding herds there is a gradual accumulation of susceptible animals; in many herds there are a few "carriers" of Bang infection, most of which are breeding in a satisfactory manner. When the carrier becomes a true spreader and is closely associated with susceptible pregnant cows, the ground is prepared for the so-called "abortion storm".

18. The primary aim of all preventive measures is to keep spreaders and potential spreaders away from susceptible animals.

The Agglutination Test

1. When cows become infected with *Bact. abortum,* agglutinins develop in the blood. Under properly controlled conditions these aglutinins cause clumping, or agglutination, when blood serum of an infected animal is added to a saline solution containing *Bact. abortum* organisms held in suspension.

2. The exact nature of the agglutinins is unknown. They are regarded by many as defense products, but their relation to immunity is not clear, and there are some reasons for doubting the usual interpretation of their function.

3. Following infection, the rapidity and extent of the development of agglutinins is an index to the *sensitiveness* of the individual to the infection. In general terms the more prompt and profuse the elaboration of agglutinins, the more likely is abortion to take place.

4. The essential defense products which determine immunity are probably much more tardy in their appearance than are the agglutinins.

5. In animals which have reached sexual maturity the agglutinin content of the blood is sufficiently reliable in detecting *Bact. abortum* infection to be of great value.

6. The agglutination test is not a reliable index to *Bact. abortum* infection in calves. This is because agglutinin absorption may take place from the milk of a reacting dam directly into the blood stream of the calf, thus causing it to react. Also, the calf rarely becomes infected in the true sense of the term.

7. When blood serum samples are set with *Bact. abortum* antigen in dilutions of 1–20, 1–40, 1–80, 1–160, 1–320, 1–640, respectively, the following essential facts appear:

A. The blood of the fetus shows no reaction in any dilution.

B. The blood of the calf shows no reaction in any dilution until the animal takes colostrum.

C. After the calf takes colostrum which is high in antibody content the blood will react in a few hours.

D. From day to day, from week to week, there may be rather violent fluctuations in the agglutinin content of the calf's blood.

E. Soon after the calf is weaned the blood ceases to agglutinate *Bact. abortum* antigen, or agglutinates it in the lower dilutions only (1–20, 1–40).

F. In non-infected heifers and cows the agglutination reaction fluctuates in the dilutions up to, and including, 1–40. This is due to normal, or group agglutinins.

G. In infected heifers and cows the agglutination reaction fluctuates among the dilutions above 1–40.

H. A reaction of 1–40, and even one at 1–80, comes in the doubtful group. It is desirable to retest cows that react in these dilutions. A reaction at 1–80 is usually specific. One at 1–40 often is not.

I. A series of tests gives more information than a single test. One test does not affect the accuracy of subsequent ones.

J. Following infection of pregnant cows by feeding, the agglutination curve usually starts upward in from two to six weeks. The rise tends to be rapid, but may be gradual. Violent fluctuations between the dilutions which denote infection and those which denote freedom from infection are not common.

K. Following infection brought about by intravenous or subcutaneous inoculations the rise in the agglutinin titre of the blood is more rapid than is observed following infection by feeding.

L. Following the initial infection, as indicated by agglutinations at 1–80 or above, most cows and heifers eliminate *Bact. abortum* with the uterine discharges at the next parturition. Whether the animals abort or calve depends largely on whether infection occurs early or late in gestation. Individual resistance is also a determining factor.

M. Subsequent to parturition which has followed the initial high reaction in an individual, the agglutination curve will usually remain high for an indefinite period (a year or several years). In some animals the curve declines, in which instances the decline is usually gradual.

N. The prolonged high reaction usually is associated with localization of *Bact. abortum* in the udder or lymph glands, rarely in the genital tract. Many cows reacting thus are satisfactory breeders.

O. The status of the cow, naturally infected, whose agglutination curve descends to normal, and is thus maintained, is not well determined. Certainly there is a degree of immunity, or resistance, developed, but whether the majority of such animals are *Bact. abortum* carriers is yet to be determined. Our work to date indicates that they are not, but our data on this point are meager.

PRACTICAL APPLICATION OF THE AGGLUTINATION TEST

1. As a means of *herd* diagnosis. If we know that Bang infection is active in the herd we know that it will be responsible for many abortions and for associated ills. Regardless of whether there are other infections present, we know that the ills due to Bang abortion disease will continue, periodically at least, as long as our efforts are limited to genital hygiene only.

If we know that Bang abortion disease is not present in a herd, we have more assurance that purely local genital infection is to be dealt with. We know also that we have a unit which is highly susceptible to *Bact. abortum* infection, and is to be guarded against this infection when purchases are made.

2. In protecting sound herds against the dangers due to purchase of recruits which are carrying Bang infection. (Testing all purchases.) High reactors are practically all carriers. Any

carrier may become a spreader. Any spreader is capable of infecting susceptible pregnant cows through channels other than the genital tract.

3. In protecting clean purchased animals from associating with infected animals (especially the long-time reactors) which may be in the home herd. The status of the home herd must be known, and the status of the purchased animals must be known. It is as disastrous to place clean, susceptible animals in an infected herd as it is to place infected animals in a clean herd. Exchange should take place between sound herd and sound herd, or between infected herd and infected herd. Mature pregnant cows with records of previous infection are the safest venture, where recruits are to be added to an infected herd.

4. In diagnosing *Bact. abortum* infection in bulls, those either with satisfactory or unsatisfactory breeding records.

5. In detecting the few infected animals in a herd recently infected.

6. In periodic herd tests enabling the owner better to know the status of the individual cows, and to detect a vantage point from which the final move toward a clean herd can be made.

7. In enabling the breeder to utilize his maternity stalls to better advantage, giving preference to infected cows, so as to prevent, so far as possible, new infections.

8. In making the differentiations on the occasional farm where the two unit system (infected cows and those which are clean) can be employed from the beginning.

9. In detecting the occasional infected heifer when an effort is being made to establish a clean breeding unit, through segregation of the heifers as they reach breeding age.

In conclusion let it be said that each herd is an individual problem in itself. The method of attack in freeing any herd from *Bact. abortum* infection will be determined by conditions which are peculiar to that herd. The foregoing suggestions *are not offered as a substitute for obvious attentions which the individual cow may require.* If a placenta needs to be removed there is no safe substitute for its removal; if a cow is sterile there is no substitute for a thorough examination to learn the cause; and there is no substitute for the constant veterinary service which is involved in these operations. But in those herds in which the "ounce of prevention" of *Bact. abortum* infection can be substituted for the "pound of cure," breeding efficiency will be at its best; in those so situated that *Bact. abortum* infection must be tolerated, there will be continuous, or intermittent, breeding trouble due to it, and our clients should be acquainted with this truth.

BANG ABORTION DISEASE IN CATTLE*

R. R. BIRCH AND H. L. GILMAN
Veterinary Experiment Station

Infectious abortion, or, more correctly, Bang abortion disease, is probably the most prevalent and destructive malady with which the modern cattle breeder has to deal. Because of its peculiar nature and of successive disappointments which have attended various single remedies alleged to prevent or cure it, there has grown up a belief among cattlemen that nothing is known regarding the diease. As a matter of fact, definite knowledge regarding it has developed rapidly in recent years, and it is only by understanding and employing the truths that have been given us that we may hope to control this scourge of our cattle herds.

This paper is an attempt to sift out and to present essential and well-verified facts regarding Bang abortion disease, and to make suggestions that will aid in preventing or in handling it. While much of the material represents collective efforts of scores of investigators, it is, nevertheless, true that we have been careful to verify or establish by our work the essential statements that are included. Enough is known regarding Bang abortion disease to enable us to control it, if only the facts regarding it can be employed consistently and intelligently.

ESSENTIAL FACTS REGARDING BANG ABORTION DISEASE

CAUSE

The cause of Bang abortion disease is *Bacterium abortum*, a germ discovered in 1895 by Professor Bang of Denmark. He recovered it from fetuses and fetal membranes of aborting cows, and went far toward establishing it as the cause of the abortions. His work has subsequently been supplemented and confirmed by scores of investigators representing practically all civilized countries. *Bacterium abortum* is not the only germ capable of producing abortion in cattle, but it is active in most herds in which abortions are frequent. In the many herds we have examined in New York, we have in but one instance failed to demonstrate severe *Bacterium abortum* infection whenever there were frequent abortions. In this, our work agrees closely with that of investigators in other States.

NATURE

Bang abortion disease is a specific infection which should be classed with systemic maladies, such as tuberculosis, and not with the various infections which cause local inflammation and pus formation in wounds. In the infected cow the germs exist in the fetal membranes within the pregnant uterus, in the udder, the

* Published as Cornell Extension Bulletin 137, April, 1926.

spleen, and the lymph glands. The germs may remain in the udder and the lymph glands for months or years, but the tendency is for them to disappear from the uterus in a few weeks after the infected cow aborts or calves. They cause abortion by attacking the fetal membranes on which the life of the fetus depends.

ESCAPE OF THE GERMS FROM THE BODY OF THE INFECTED COW

When the infected cow aborts, and in many instances when she calves at full term, the germs are eliminated in enormous numbers in the fetal membranes and in the discharges from the uterus. Many infected cows eliminate the germs in the milk, either continuously or at intervals covering months or years. Bulls do not often become infected; but some of those that do, eliminate the germs in the seminal fluid. The germs will sometimes pass unharmed through the entire digestive tracts of calves and mature cattle. Therefore, calves drinking milk from infected cows aid in spreading the germs, although the calves themselves seldom become infected.

THE GERMS OUTSIDE THE ANIMAL BODY

Outside the bodies of cattle the Bang organism will grow readily in test tubes where especially favorable conditions are provided, but it is doubtful whether it multiplies habitually in external nature. It will, though, live for a considerable time in discharges from the genitals of infected cows. Dampness and darkness tend to preserve it, while sunlight and drying operate to destroy it. It is killed readily by heat and by ordinary disinfectants, such as bichloride of mercury and compound cresol solution.

HOW COWS BECOME INFECTED

So far as we know all new infections originate in "spreaders,"—that is, in animals that are giving off the germs in their discharges. These discharges (exudate from the uterus, or the milk) soil bedding, hay, or pasture, and are thus taken up by pregnant cows in the feed. Cows may also become infected by licking aborted fetuses, newborn calves of infected cows, fetal membranes, or the external genitals of other cows that have aborted. From the digestive tract the germs are transferred through the blood or lymph to the pregnant uterus, where they localize in the fetal membranes, invade the fetus, and ultimately cause its death and expulsion. Artificial cultures of the Bang organism fed to pregnant cows cause a high percentage of them to abort.

THE BULL AS A "SPREADER"

It is possible for the bull to introduce the Bang organism into the genital tracts of cows in two ways: first, by serving a cow which is discharging the germs from her genitals, and soon afterward serving another one that is clean; secondly, by harboring the germs

in the testicles or in the various passages through which the seminal fluid must flow before it is discharged into the vagina of the cow. The former method of infection is rare, because the Bang organism does not as a rule remain in the discharges from the uterus of a cow long after she aborts or calves. Nevertheless, it is a wise precaution to permit an infected cow to go over two periods of heat before rebreeding, and, as a practice, to douche the sheath of the bull with a 0.25 per cent Lugol's solution before and after each service. Exceptionally, a bull will acquire deep-seated *Bacterium abortum* infection of the genitals which causes him to discharge the germs with the seminal fluid at each service. The fact that this type of infection is rare does not detract from its seriousness, should it occur. Douching the external genitals has no effect whatever on infection of this nature. When it is discovered, the bull should be retired from service permanently.

In this connection it should be emphasized that occasionally infections entirely apart from the Bang organism are carried by the bull. These may be transferred by the bull from cow to cow during services that take place near the same time, or they may become deep-seated, either in the cow or the bull, and ultimately cause sterility. While no investigation of breeding trouble is complete without consideration of the bull, he does not as a rule play a prominent part in the spread of Bang abortion disease. Other highly injurious infections which he at times transfers are far less prevalent than the one caused by *Bacterium abortum.*

INDIVIDUAL RESISTANCE

Most pregnant cows that previously have not been infected will, on exposure, contract Bang abortion disease. Heifers seem most susceptible. Some females resist the infection altogether; and others resist it to a degree which enables them to carry their calves to full term, even though the fetal membranes harbor the organisms. Bulls usually resist the infection, and there is some evidence that non-pregnant females are much less susceptible than are pregnant ones. The fetal membranes, present only in the pregnant cow, furnish ideal soil for the growth of the organisms.

Heifer calves that have not reached sexual maturity seldom become infected, even when they are allowed to associate freely with "spreaders" and to use them for nurse cows. Calves reared in such surroundings, if removed to clean quarters as they near sexual maturity, do not, as a rule, acquire the infection; most of them become safe breeders unless they are exposed to the infection near or after breeding time. This is a well-verified fact and an exceedingly important one, for it enables the breeder who takes advantage of it to build up a clean herd out of a badly infected one and, at the same time, to preserve blood lines which are highly prized.

IMMUNITY, OR TOLERANCE, DEVELOPED BY NATURAL INFECTION

A fairly effective immunity, or tolerance, which protects most cows against subsequent Bang infection resulting in abortion, is developed as a result of natural infection. Thus cows that have aborted once, or more rarely twice, as a result of Bang infection, provided they are not rendered sterile, subsequently resist the infection to such an extent that they calve at full term. Apparently some recover entirely from the effects of the disease, while others harbor the organisms in the udder or lymph glands indefinitely. Some cows become infected, calve at full term, and develop immunity as a result. A few individuals develop no immunity whatever and abort year after year. Whether each abortion in a series of this kind is due to Bang infection or to other organisms which cause chronic inflammation of various parts of the genital tract has not been fully determined.

ARTIFICIAL IMMUNITY

It has been shown that the immunity which develops as a result of natural infection plays a very important part in protecting females against subsequent exposure. Naturally, attempts have been made to produce this immunity with artificial cultures. Vaccines consisting of live virulent cultures of the Bang organism and bacterins consisting of killed cultures of the same germ are the products employed. The vaccines are not given to pregnant cows but are administered some months before breeding time in the hope of bringing about immunity before the animals become pregnant. The bacterins are administered either before or after breeding, or at both times.

Vaccines, no doubt, bring about immunity in some individuals, but the real question is whether the good they do outweighs the harm that attends their use. While experiments so far conducted do not answer this question with entire satisfaction, it can be said that harm, as well as benefit, attends their use; that failures to produce immunity with them are frequent but are not advertised; and that in their present state of development they are by no means a safe and reliable resort.

Following a particularly disastrous year in which abortions and allied troubles are frequent, the breeder, in despair, is ready to try anything that holds out the least hope. He frequently tries vaccine. Because there are no longer large numbers of susceptible animals in the herd anyhow, the abortion rate declines, the vaccine receives credit for the decline, and the breeder in all sincerity may pass the good news to his neighbor or write a testimonial expounding the virtues of vaccines.

The bacterins used, as we have already explained, are killed cultures of the Bang organism. No great danger attends their use and they give a degree of protection in some individuals. Failures attending their use are frequent, and as now prepared and

used they are not uniformly dependable. For several months following their use bacterins, and vaccines as well, cause cows to react to the blood tests which we shall describe presently, thus rendering the tests valueless in identifying infected animals.

SYMPTOMS

Following infection of the pregnant female it usually requires from 30 to 120 days for symptoms to appear. While there is at times a slightly perceptible loss in condition, the infection does not at first materially affect the general health of the animal. The outstanding manifestations of the disease consist of death of the immature fetus and its expulsion before term. Expulsion of the immature fetus is usually preceded by the same outward signs that signal the approach of normal birth. The udder enlarges, the vulva becomes swollen, ligaments adjacent to the external genitals relax, and usually a discharge precedes the expulsion of the fetus. Before these changes occur, there have for weeks been taking place in the fetal membranes unseen changes that prevent them from supplying to the fetus elements necessary for life and development.

Cows that abort early in pregnancy usually do not retain the fetal membranes, but, as pregnancy advances, retained membranes are most frequent. Following abortion or calving, especially when the fetal membranes are retained, pus-producing germs gain entrance into the uterus and cause severe and lasting inflammation which may result in temporary or incurable sterility or may even bring about general symptoms which threaten the life of the animal. In the absence of these complications or in the event of their skillful treatment, most cows breed subsequently and carry their calves full term.

DIAGNOSIS

There is no substitute for a correct diagnosis in handling disease. We cannot hope to cope successfully with a transmissible malady unless we understand something of its cause, its manner of transmission, and the means by which it may be diagnosed. Bang abortion disease may be diagnosed in three ways:

First, by cultures made directly from a fetus recently aborted.

Secondly, by inoculation of guinea pigs with milk or with discharges from the uterus of suspected cows. (*Bacterium abortum* produces characteristic changes in the spleen of guinea pigs.)

Thirdly, by blood tests.

In skilled hands a positive diagnosis made by any of these methods is conclusive, but in actual practice the blood tests are simplest and most convenient and have the distinct advantage of detecting many infected animals before other indications of abortion appear. The agglutination test is the one most commonly applied.

The Agglutination Test

If we grow cultures of the Bang organism and then shake the germs up in a weak salt solution, a cloudy fluid is produced. If we drop into this fluid a certain quantity of blood serum from a cow that is free from *Bacterium abortum* infection and let the mixture stand over night under properly controlled temperature conditions, it remains unchanged; if we drop into another tube of the same cloudy fluid a like quantity of blood serum from a cow that is harboring *Bacterium abortum* infection and allow it to stand over night in the same manner, the fluid will be clear in the morning and the germs will appear in clumps which adhere to the bottom of the tube. This clumping is called agglutination.

The relative quantity of blood serum required to cause this clumping determines the probability of infection. It is customary in making the tests to use in the first test tube one part of the blood serum to be tested to twenty of the cloudy germ-laden fluid which has been described, a 1–20 dilution. Other tubes are prepared using dilutions of 1–40, 1–80, 1–160, 1–320, and 1–640, respectively. The blood serum of normal cows will cause no clumping at all or the clumping will appear only in the lower dilutions (1–20 and 1–40). The blood serum of infected cows usually causes clumping in dilutions of 1–80 or above. While it is always advisable to retest cows that react as high as 1–80, most of them will be found on subsequent investigation to be infected. Doubtful cases may be tested again and again, as one test does not interfere with another. A few infected cows fail for a time at least to react, but a reaction in the higher dilutions (1–160 or above) is almost always associated with *Bacterium abortum* infection.

A reaction to the agglutination test means that the cow is, or recently has been, infected. It does not mean necessarily that she will abort, for infection may take place late in pregnancy or the cow may have sufficient resistance to prevent actual abortion. Following infection and consequent reaction, the fetal membranes of most cows contain *Bacterium abortum* when the next calf is expelled, whether it is dead or alive. In subsequent pregnancies approximately 20 per cent of the reacting cows are "spreaders" from the uterus at the time when they abort or calve. These old reactors continue to harbor the infection in the udder and lymph glands, but the uterus tends strongly to free itself of the infection and to repel subsequent invasions.

The following chart illustrates some of the more important points regarding the agglutination test.

AGGLUTINATION RECORD OF A GRADE HOLSTEIN HEIFER *

Date blood sample was drawn			Dilution						Remarks
			20	40	80	160	320	640	
1923	March	15	+†	—	—	—	—	—	
	June	17	+	+	—	—	—	—	
	July	16	+	+	—	—	—	—	
	August	15	+	P	—	—	—	—	
	September	15	+	+	—	—	—	—	
	October	16	+	—	—	—	—	—	
1924	February	18	+	—	—	—	—	—	
	March	15	—	—	—	—	—	—	March 18 — Bred
	April	17	—	—	—	—	—	—	
	May	16	—	—	—	—	—	—	May 26 — Infection period began
	June	2	—	—	—	—	—	—	
		9	+	+	—	—	—	—	
		16	+	+	P	—	—	—	
		25	+	+	+	+	—	—	
		30	+	+	+	+	+	+	
	July	7	+	+	+	+	+	—	
		14	+	+	+	—	—	—	
		21	+	+	+	+	+	+	
		28	+	+	+	+	+	P	
	August	11	+	+	+	+	+	+	
		18	+	+	+	+	+	+	
		25	+	+	+	+	+	+	August 27—Infection period ended
	September	1	+	+	+	+	+	+	
		8	+	+	+	+	+	+	Cow aborted. Retained placenta
		15	+	+	+	+	+	+	
		22	+	+	+	+	+	+	Fetus, milk, and placenta contained *Bacterium abortum*
		29	+	+	+	+	+	+	
	October	6	+	+	+	+	+	+	
		13	+	+	+	+	+	+	
		20	+	+	+	+	+	+	
		27	+	+	+	+	+	+	
	November	3	+	+	+	+	+	+	
		10	+	+	+	+	+	+	
		17	+	+	+	+	—	—	
	December	1	+	+	+	+	P	—	

* This heifer was born March 3, 1923.
† + = agglutination; — = no agglutination; P = partial agglutination.

The blood of this heifer, born March 3, 1923, was tested each month. Normally she reacted at 1–40 or below, as the monthly records show. She was bred on March 18, 1924. On May 25, 1924, and thereafter three times weekly for three months, she had sprinkled on her feed a small quantity of fluid containing *Bacterium abortum*. On June 25, about a month after the first infectious material was fed, she was reacting at 1–160. The high reaction persists, though it is not exactly the same month after month. The cow aborted on August 27, 1924, about three months after the first infectious material was fed; the fetus, the fetal membranes, and the milk were loaded with *Bacterium abortum*, the germ which caused the abortion.

This heifer was one of the nine which were bred to the same bull and which were infected in the same manner. Eight became reactors, six aborted, one calved prematurely, and two calved at full term. The six aborters and the one that calved at full term were "spreaders" from the uterus at the time the calf was expelled. Seven other heifers from the same source, bred to the same bull, kept in a separate part of the same barn, but not fed

Bacterium abortum cultures, all failed to develop reactions and all calved at full term. Careful examination of the fetal membranes was made and *Bacterium abortum* could not be found.

Let us return to the heifer included in the chart. Her uterus was cleaned up thoroughly after she aborted, she is again pregnant and is a high reactor, 1–160. What of her breeding future? If she behaves as most cows we have observed under like circumstances, she will carry the calf and subsequent ones to full term. There is about one chance in five that her fetal membranes will contain *Bacterium abortum* when she calves. If she is similar to other cows we have examined, her high reaction is due to localization of *Bacterium abortum* in her udder or lymph glands. This infection may remain for years, in which case the high reaction will continue; it may, however, die out, and in that case the high reaction will ultimately disappear and complete recovery will take place. Whether the infection in the udder and the lymph glands persists or not, the cow's uterus will tend strongly to resist infection in subsequent pregnancies. Placed in a bady infected herd she will be far more likely to breed safely than will cows that have never been infected.

If we were to test this cow during her second pregnancy for the first time and examine her for pregnancy, we would find that she is pregnant and a high reactor. If we did not know that she had been infected in a previous pregnancy and had developed resistance as a result, we might assume that she is at the present time in great danger of aborting. A single high reaction in a heifer in first pregnancy signifies that she is likely to abort and is almost certain to be a dangerous "spreader" when the calf is expelled, whether it is dead or alive. A single test and high reaction in an older cow *recently infected* means the same thing, but we have no way of knowing from a single test of the older cow whether the reaction indicates recent infection with impending abortion or infection of long standing which has caused the cow to develop resistance sufficient to prevent abortion. Thus, in older cows, a number of tests gives more information about the probable breeding future of an individual than does a single test; but, in any event, a high reactor should be handled on the assumption that she is exceedingly dangerous to her associates at the time when she aborts or calves. The milk may aid in spreading the infection if it is carelessly handled.

The agglutination test is not perfect in its operation. It fails to detect an occasional cow that is infected, and a few fall into the suspicious class; but a high reaction means that the individual involved is, or recently has been, infected with the Bang organism. The suspicious cases may be retested as often as is necessary to learn their true status. Carefully employed and used by those who understand it, the agglutination test gives information of the greatest value in protecting herds against Bang abortion disease and in weeding out animals which are likely to infect their associates.

SUMMARY

In order that the reasons for the choice of methods of control may be apparent, let us review briefly a typical history of *Bacterium abortum* infection both in individual cows and in a herd. The heifers rarely become infected before breeding age and relatively few react before becoming pregnant. If exposure takes place, particularly during pregnancy, a reaction develops; the heifer aborts or calves, and eliminates large numbers of *Bacterium abortum* in the fetal membranes, in the uterine discharges, and sometimes in the milk. The fetal membranes in many instances are retained, and unless skillful local treatment is applied, chronic infection with various pus-producing organisms develops, leading in many cases to sterility. If given appropriate treatment, most females breed subsequently and carry their calves to full term. They possess a degree of immunity or resistance which in most cows prevents further abortion. Because of localization of the organisms in the udder or the lymph glands, many such animals continue to react indefinitely. Some eliminate *Bacterium abortum* in the milk for months or years, and a few that abort a second time or that calve at full term eliminate the organisms in the fetal membranes and the uterine discharges.

Bacterium abortum infection may invade the herd rapidly or slowly. If it becomes well established, ultimately most susceptible cows become infected and many of them abort as a result. Some resist the infection altogether; others resist it to a degree that prevents actual abortion, even though the pregnant uterus is invaded; some become sterile; and a few even die as a result of uterine infection caused by secondary invasion with other germs. In most herds that are well handled, the majority of the females emerge from the first attack as fairly satisfactory breeders. Many, however, continue to eliminate the organisms in the milk, occasionally in the uterine discharges, and thus, at times, become dangerous to susceptible associates. In this manner the infection may smoulder for years in a herd without being outwardly manifest. In the absence of actual abortion, the breeder is likely to assume that his herd is free from infections which cause abortion. In the meantime there is a gradual accumulation of susceptible stock. Heifers grow to breeding maturity and purchased recruits are added to the herd. Then, when one of the old reactors becomes a true "spreader" at calving time, the stage is set for another siege with the disease. *Open infection and susceptible pregnant cows* in the same herd at the same time usually bring about disaster. A herd consisting entirely of reactors may be a fairly satisfactory breeding unit. One consisting of clean animals, which for the most part are susceptible, is far more satisfactory. One consisting of individuals from both groups will usually be a source of loss and disappointment to the owner.

THE HANDLING OF BANG ABORTION DISEASE

In handling Bang abortion disease the cattle breeder encounters two problems: one is to prevent the infection from entering his

herd, or, once introduced, to prevent its spread from animal to animal; the other is to provide appropriate treatment for animals that unavoidably become infected. It is always highly profitable to substitute prevention for cure, but, should prevention fail, the kind of treatment applied to the individual cow just after she aborts or calves determines in a large measure her breeding future.

When new infections occur, susceptible animals have come in contact with "spreaders" or with material soiled by their discharges. Susceptible animals are sexually mature cattle, particularly pregnant females, that previously have not been infected. "Spreaders" are sexually mature infected females that are giving off *Bacterium abortum* in the discharges from the uterus or in the milk; bulls with deep-seated genital infection resulting in discharge of the germs in the seminal fluid; and calves that drink the milk of infected cows and eliminate the organisms in the feces.

PROTECTING THE SOUND HERD

Most clean herds become infected through the introduction into the herd of infected animals, more often females. The infected pregnant cow which becomes a "spreader" when the calf is expelled and the infected cow which recently has aborted or calved, are particularly dangerous. A breeder with a clean herd should avoid speculative purchases, confining recruits to such animals as actually are needed to improve his herd. In protecting a clean herd against Bang abortion disease it is important to know the following points regarding each female purchased:

1. Her age and previous breeding history.
2. Whether she is pregnant, and, if so, the date of service and to what bull.
3. Whether any part of her genital tract is visibly diseased.
4. Whether she comes from a prolific herd. (Evidence should exist in the form of young stock.)
5. Whether she is a reactor to the agglutination test for Bang abortion disease, and, if possible, whether she comes from a herd in which other reactors exist.

Careful inquiry should be made into the breeding history of each bull that is to enter a clean herd, and he should be shown by physical examination and the agglutination test to be free from infection likely to be transmitted to the cows he serves.

A *sixty*-day retest of all purchased animals is a wise precaution, and, if possible, the animals should be kept away from the breeding herd until this final test is passed safely.

A perfect breeding history for an individual cow does not give complete assurance that she is not a "carrier" of detectable Bang infection, and a menace to susceptible animals with which she comes in contact. In most instances the agglutination test will supply this information.

Where herds of considerable value are involved and where the breeder has access to the services of a thoroughly qualified veterinarian, the latter should be employed to aid in selecting breeding

animals. Too often the veterinarian is called after the disaster occurs rather than as an aid in preventing it.

It always is of great advantage for the breeder to know the status of his own herd as regards *Bacterium abortum* infection, *for it is as disastrous to add a given number of clean cows to an infected herd as it is to add an infected individual to a herd containing the same number of clean cows.* If it is necessary to purchase cows to add to a herd consisting mostly of reactors, mature pregnant cows with a history of previous infection are the safest venture. In other words, so far as Bang abortion disease is concerned, transfer of breeding stock should take place between *infected herd and infected herd* or between *sound herd and sound herd.* This minimizes the danger of placing susceptible breeders in contact with "spreaders," always an exceedingly dangerous practice. Once a herd is clean, a semi-annual or annual test is a wise precaution.

HANDLING THE INFECTED HERD

While the methods of spread of Bang abortion disease are essentially the same in all herds, each herd constitutes a problem in itself. The object to be attained in each instance is the same, but the method of procedure is determined entirely by the circumstances which surround the individual herd. The number and value of the animals, the plan of herd management, the number of animals infected, the number of maternity stalls, the availability and the efficiency of veterinary service, the reliability and the alertness of those in direct charge of the cattle, all are factors which must determine the methods of attack.

Before we suggest various plans of control it is necessary to direct attention to certain principles that apply, regardless of the plan which finally is adopted.

Correct diagnosis is necessary. We need to know with what disease we are dealing. Is it actually Bang abortion disease? How many cows are infected? What is the status of each cow of breeding age with regard to *Bacterium abortum* infection and with regard to other conditions which interfere with breeding? Is there real evidence that the bull is a factor in the spread of the disease? The answers to these questions as they apply to the herd or to the individual cow are procurable, and they form the basis of any method of control that is to attain lasting success.

Veterinary service is essential. The breeder can scarcely hope to cope successfully with his problem without competent veterinary service. In the first place, correct diagnosis and appraisal of actual conditions existing in the herd and in the individuals, can be made only by men with intimate knowledge of disease. Then, there is the constant need for a trained man close at hand to perform services which are beyond the skill of the breeder. Examinations to determine pregnancy or sterility, removal of retained fetal membranes, treatment to correct conditions that delay breeding or cause permanent sterility, drawing blood samples for agglutination tests, demonstration of a suitable method of cleansing the sheath of the bull before and after each service, and examination of the bull

with a poor breeding record, are just a few of the duties which may be required in any breeding herd and which demand the services of a specially trained man. In addition, the truly competent veterinarian offers indispensable guidance regarding certain details, some seemingly trivial but actually of the very greatest importance.

There must be close attention to "spreaders" and to the method of detecting them, in order that they may not infect their associates. Until it is known positively that a particular abortion is not due to the Bang organism, precautions should be taken on the assumption that it is the cause. These precautions consist of the immediate segregation of aborters and prospective aborters; burning or burying the fetus, the fetal membranes, and the contaminated litter; and thorough disinfection of the stall vacated by the aborting cow. Calves that nurse cows not known to be clean are to be regarded temporarily as "spreaders" and not allowed the freedom of the dairy barn.

Wherever possible maternity stalls should be employed. If each cow could be entirely away from her associates during the few days previous to aborting or calving and during the four weeks following expulsion of the calf, this in itself would often act as an effective check on the spread of *Bacterium abortum* infection in a herd. The maternity stall needs to be roomy and comfortable. It must be so situated that thorough disinfection can conveniently be carried out and that the removal of manure does not contaminate the quarters of clean animals. When the stall is vacated by one cow, it needs to be thoroughly cleaned and disinfected before it is used again. Otherwise it may act as a center of infection, threatening every individual cow that enters it.

Appropriate treatment should be applied promptly to cows that abort, retain their fetal membranes, or develop any of the numerous abnormalities that interfere with breeding, or else the animals should be sold for beef as soon as their condition will permit. No two cases develop in exactly the same manner, and each case is best handled under the guidance of a competent veterinarian. As a rule, fetal membranes retained more than six hours cannot safely be removed before an interval of approximately two days. Far more harm is done by removing them too soon or imperfectly than is done by leaving them entirely alone. The more effective our preventive measures become, the less treatment individuals in the herd will require, and the more satisfactory will be the yield in milk and calves.

Reliable men must be in direct charge of the cattle if there is to be any real attempt to rid a herd of *Bacterium abortum* infection. Men low in intelligence, or irresponsible, may observe under compulsion certain precautions which secretly they regard as unnecessary, but they relax all vigilance when they are left alone with the cattle. Money paid for a competent herdsman is well invested.

Careful breeding histories of all cattle are essential, and, if supplemented with periodic agglutination tests and physical examinations as required, they serve to reveal the true status of each animal.

Bulls should be free of genital infection, and in doubtful cases should be retired from service until all doubt regarding their fitness to breed is removed. Cows should be bred only when there is reasonable assurance that their genitals are normal and free from infection. The practice of breeding a "shy breeder" to several bulls cannot be too strongly condemned.

Specifics for Bang abortion disease have no proved value, and they cannot be made to play a part in any plan that succeeds. Most remedies of this character are harmless to the individual animal, but they do great harm in holding out false hopes, and thus delaying or preventing the application of measures which really are effective. Usually these remedies are given when the abortion rate is so high that a decline is in prospect anyhow, and the breeder who confines his observations to a single herd sometimes credits the decline to the remedy. Only when there again accumulate considerable numbers of susceptible animals which become infected and abort in spite of the remedy, does the breeder lose faith in it, and try another. Various simple remedies, such as carbolic acid, methylene blue, potassium iodide, numerous "mineral mixtures," and the like, have appeared and gone their way, mere bubbles on the surface of a complex problem. Some day there may be an effective specific for Bang abortion disease, but when it appears its value will be made known by those who have no financial interest in it.

SUGGESTIONS REGARDING INDIVIDUAL METHODS OF CONTROL

The Two-Unit System of Herd Management

The two-unit system of herd management consists of an initial agglutination test on all individuals of breeding age and subsequent maintenance of reactors and nonreactors in units entirely separate. In the nonreacting division the initial test is followed by periodic tests to detect reactors not revealed originally. In beginning, especially with herds badly infected, these retests need to be made rather frequently, but, as the clean unit becomes established, semiannual or even annual tests may be sufficient. Great care needs to be practiced in watching for prospective aborters in the nonreacting unit, for an occasional abortion occurs which the agglutination test fails to detect. Suspicious animals should be kept apart temporarily and followed with the agglutination test until their true status in relation to *Bacterium abortum* infection can be learned. Many modifications of the absolute two-unit system suggest themselves, for as yet there are relatively few farms so equipped as to employ it to advantage.

Selling Bang Abortion Disease Out of a Herd

The selling of Bang abortion disease out of a herd is really a modification of the two-unit system just described, the aim being to sell the infected animals instead of retaining them in separate quarters. As ordinarily practiced it consists merely of selling

cows that actually abort and purchasing others to replace them. It rarely succeeds when thus limited. Because of the fact that there remain in the herd certain "carriers," some of which may never have aborted, and because cattle purchased or young stock raised may be highly susceptible, the two elements which always cause trouble — *Bacterium abortum* infection and susceptible cows — still remain in the herd. Many of the cows sold might otherwise remain in the herd, develop resistance, and become satisfactory breeders; in many instances the purchased cows are highly susceptible, and merely add fuel to the flame. At other times they are infected before being purchased, in which case they possess no advantage over the animals sold.

There are circumstances under which the selling of infected animals for beef is advisable, but their selection should be based on a series of agglutination tests supplemented by the breeding record. This plan gives reasonable assurance of removing all prospective "spreaders," especially if retests are faithfully employed. When there is a limited number of reactors, some of which are of doubtful value, and a considerable number of clean cattle, this procedure has much to recommend it. At present there is no legitimate outlet for reacting animals except as they are sold for beef, but, in reality, the long-time reactors that are pregnant are good property for the man who is making no effort to free his herd of the infection.

Limiting Spread of Bang Infection When It First Enters a Herd

The first outward manifestation usually consists of an abortion. The breeder too often attributes it to accidental injury and does not heed the first warning. As a matter of fact, abortions due to accidental injury are very rare, and the only safe procedure is to take the precautions that would be observed in case the abortion were known to be caused by *Bacterium abortum* infection, namely, immediate segregation of the animal; scrupulous care in disposing of fetus, fetal membranes, and uterine discharges so that they will not infect other animals; and immediate and thorough disinfection of the stall, floor, or place in the pasture which has become contaminated. A blood sample from the aborting cow should be drawn and the agglutination test applied. If the cow does not react, a second test should be applied as a precaution in about thirty days. If it likewise fails to reveal reaction, there is reasonable assurance that *Bacterium abortum* infection did not cause the abortion.

If the cow proves to be a reactor, it is often a good plan to test all individuals in the herd which are of breeding age, including the bull, in order to learn the extent of the infection and which individuals are involved. If the infection is limited to a very few individuals, none of which are of exceptional value, they should be sold for beef or segregated at once. If the herd involved is of considerable size it may be the breeder's last chance

to get a strangle hold on the infection without heavy expense. Following the first test a series of retests may be made in order to detect and to remove tardy reactors. Always a suspicious case should be followed with the test until she falls definitely into the infected or the sound group.

Growing a Sound Herd from the Young Stock

In handling Bang abortion disease we have advantages not associated with tuberculosis — we may retest as often as seems desirable and one test does not interfere with later ones; also, calves only exceptionally contract permanent infection when allowed to associate freely with reactors and to take milk from them. This latter fact makes possible a method of attack which may be employed in some breeding establishments not equipped to take complete advantage of the two-unit system.

A herd may contain a considerable number of reactors, the removal of which, at one time and without replacements, would too seriously limit the immediate income derived from it. The reactors, many of which have been infected for some time, may be fairly satisfactory breeders and milk producers but dangerous, particularly at calving time, to their associates. In such herds the heifer calves, after weaning, may be kept apart from the main herd, bred, and not returned to the herd until after they have calved for the first time. An agglutination test applied a short time before the heifers are bred and retests made during pregnancy, will aid in detecting the occasional heifer which may carry infection from calfhood or become infected otherwise.

When the heifers thus reared are ready as replacements, the main herd may be tested; the reactors sold for beef, or the more choice animals segregated; and the barn thoroughly cleaned and disinfected. Following this, retests should be applied to detect new infections or delayed reactors, and the individuals should be observed closely for signs of impending abortions.

Partial or Imperfect Segregation of Infected Animals

Some breeders whose establishments are not equipped to provide complete segregation for the young stock as just described may still be able through careful work to limit the spread of the infection in their herds. The plan centers around extensive and judicious use of the maternity stall. If each cow can be placed in a maternity stall during the few days prior to calving or aborting and during the three or four weeks following, and not returned to the herd until all abnormal discharge from the genitals ceases, this practice alone will greatly limit the spread of *Bacterium abortum* infection in a herd. Even placing the reactors together in one section of a barn somewhat apart from the sound animals and caring for them last each morning and evening may limit the number of infections, provided careful men are in charge. When maternity stalls are too few to accommodate all the cows for the

time required, preference should be given to reacting cows, for they are the ones most likely to infect their associates. We have already dwelt on the necessity for cleaning and disinfecting maternity stalls after each cow is removed.

Under circumstances which forbid the use of the agglutination test in detecting infected animals, consistent use of the maternity stall often is the best plan available. This assumes, in effect, that all cows are infected, and separates each from her associates during the interval when she is most likely to spread the infection. It limits the spread of the disease to a great extent and may, at times, succeed even for a term of years, but it still is very likely to leave infected individuals in the herd. Sooner or later a "spreader" calves or aborts in pasture, yard, or stanchion; the disease gains a new advantage; and disaster overtakes the herd.

Few breeding establishments are so equipped that a maternity stall may be occupied as long as thirty days by each cow at calving time. So the tendency, always, is for individuals still dangerous to their associates to be crowded out of the stalls and back into the herd. After all, this general plan is far surer when supplemented by the agglutination test which aids in detecting the principal sources of infection and thus enables the breeder to guard against them.

CONCLUSION

As we observed in the beginning, the plans of handling here outlined are based on established facts regarding Bang abortion disease and its manner of spreading from animal to animal. It is hoped that the breeder in reading them over will gain a truer conception of the disease itself and will thus be able to select a plan applicable to his own herd or to choose and employ certain features of the various plans suggested. While the ideal is to keep infected and sound animals entirely separate, the breeder often must make a more modest beginning, crowd the disease gradually out of the herd, and, whenever possible, employ an occasional test so as to indicate his progress or detect a vantage point from which the final move toward a clean herd can be made.

Inadequate equipment is one of the greatest obstacles in the way of control of Bang abortion disease and one which, of necessity, is slow to be corrected. In reality we are trying to meet a relatively modern situation with equipment and methods which are a throw-back from another generation. Our herds now are larger than they used to be, they are more closely housed, disease is far more prevalent, exchange of animals among herds is more frequent, and each exchange involves immeasurably greater risks. Because of these facts, Bang abortion disease has become one of the greatest limiting factors in our dairy herds, both in reproduction and in milk yield. Carefully selected animals, better equipment, better care, and more effective protection against disease, are the aims of the modern breeder, even though the herd must be smaller in order to accomplish these aims. A small herd of

choice cows, well protected, which really reproduces, is far more profitable than is a large herd that scarcely can maintain its numbers.

Those who have long experience with Bang abortion disease and who count the cost in calves, in reduced milk yield, in temporary and permanent sterility, in extra labor and inconvenience, know at last that prevention is the best way out. Many preventive measures, as we have endeavored to show, can be applied by the breeder who really understands the manner in which the infection spreads; but when these measures fail and the disease becomes established in a herd, immediate treatment of infected individuals and the ultimate elimination of the disease from the herd are best accomplished with the aid of competent veterinary service.

TECHNIQUE OF DRAWING BLOOD SAMPLES FROM CATTLE FOR THE AGGLUTINATION TEST

R. R. BIRCH

Veterinary Experiment Station

Requirements. Gallon receptacle containing disinfectant solution, and a brush for applying it. Two sixteen-gauge needles two and one-half inches long. Shallow receptacle containing disinfectant for needles. Gallon pitcher, or like receptacle, containing water for rinsing the needles. Round, neckless bottles about fifteen c.c. capacity as containers for blood samples. Labels, preferably of the type illustrated (Dennison's No. 217).

Procedure. (With the operator working on the left side.) Have an assistant place a bull-leader, or strong halter, on the cow, and draw her well forward in the stanchion, with the head slightly elevated and inclined to the right. Have another assistant place a quarter-inch choke-rope around the neck, low down, with the slip noose on top, and standing at the cow's flank, apply steady traction. This distends the jugular so that its course is visible. Apply disinfectant to the skin over the jugular at a point approximately six inches posterior to the margin of the mandible, and with a sharp blow force the needle quickly into the vein. Collect the bottle half full of blood, release traction on the choke-rope, then withdraw the needle and rinse it immediately by pouring water through it, not by plunging it into the water. Label the bottle and keep it in an upright position until the blood coagulates.

Discussion

Common errors in technique consist of placing the choke-rope too high on the neck, failing to release the choke-rope before the needle is withdrawn; using dull needles; too long delay, after the choke-rope is tightened, before inserting the needle; attempting to insert the needle before the course of the jugular is visible, or palpable; filling the bottle too full; failing to place the bottle in an upright position while the blood is coagulating; and failing to rinse the needle promptly.

There is a great temptation to work too low on the neck. In many instances distension of the jugular is more apparent immediately anterior to the choke-rope, but the skin at this point is so freely movable on the subjacent parts, that inconvenience is encountered in inserting the needle.

Some dispense with the choke-rope, and apply pressure with the thumb in the jugular furrow to distend the vein. The needle is pushed through the skin and into the vein instead of being inserted quickly with a sharp blow. This method is quite satisfactory, but cattle are more or less sensitive to a needle used in this manner, particularly when repeated samples are taken on the same individual.

Exceptionally, the course of the jugular is not plainly visible even after the choke is properly applied. A stroke or two with the brush, wet with disinfectant, serves to lay the hair and reveal the position of the vein. This failing, it is readily located by palpation.

If the needle is to be stabbed into the vein, it is best held rigidly with the base applied firmly to the index finger, as illustrated. Failure to do this will cause difficulty, both in piercing the skin and in guiding the blow.

When large numbers of samples are to be taken, labels in ribbon form, as illustrated, are a great convenience. Numbers of individuals in an entire row may be recorded consecutively on the labels before any samples are drawn, thus obviating necessity for drying the hands each time in order to number and apply the label. This method also renders errors in labeling very unlikely.

It is preferable to ship blood samples by parcels post, special delivery. They should be kept cool, but never allowed to freeze. Icing is unnecessary if the samples can be mailed and delivered promptly. The bottles should be stoppered securely, and under no circumstances should water, even in small quantities, enter the sample bottle.

Fig. 1. Convenient type of label.

Fig. 2. Manner of holding needle when it is to be stabbed into the vein.

IV. IMMUNIZING YOUNG PIGS AGAINST HOG CHOLERA

J. W. BENNER
Veterinary Experiment Station

The experiment to be described is a continued study of the subject. The first experiment was performed in 1922 [1] and repeated in 1923 [2], 1924 [3], and 1925.

Variations in the results of the yearly experiments, and those performed by others, have emphasized the necessity of continuing the study until the natural laws governing this phase of hog cholera immunology can be clearly and definitely defined. Slight changes have been made in the plan of experimentation from year to year. The principal object of this research has remained the same, i. e., to learn whether the vaccination of young pigs by the simultaneous and the virus alone methods would confer a solid and lasting immunity.

The "follow-up" method was included in the 1925 experiment for the first time primarily to compare the thrift of pigs so treated with that of pigs vaccinated by the other methods. In this experiment it consisted of protecting the pigs by serum alone until they were from nine to twelve weeks old, after which the simultaneous treatment was administered.

Pigs used in the 1925 experiment, with the exception of one litter, were of pure bred Chester White breeding. The exception was from a grade Berkshire sow and a grade Chester White boar.

In 1925 the ration consisted of two parts middlings to one part cornmeal and one part ground oats and barley, supplemented by ten per cent tankage and ten per cent alfalfa meal. The sows and pigs were hand fed twice daily at about 10 A. M. and 4 P. M. and were let to run on rye pasture part time. When the pigs were about twelve weeks old, part of the concentrates was fed from self feeders and the pigs were allowed to run on oats pasture.

Details of the causes of death of pigs that were expelled dead, that died on account of natal weakness, or were laid upon and killed by the sow during the first three or four days after farrowing, will not be given.

The sow's identification is used as the litter identification number and the individuals in each litter are numbered in numerical order, beginning with 1. Thus, Litter 4, Pig 1 is the sow's identification and her pigs are numbered 1, 2, 3, etc.

All dates given are in the year 1925 unless otherwise stated.

All pigs were dipped and scrubbed in a 0.5 per cent solution of lysol before being moved to cholera free ground.

At eight months of age each animal's immunity was tested by a deep hypodermic injection of virus into the side of the neck. At this time the individuals of the various groups were weighed and the influence of the method of vaccination on weight studied.

The terms serum and virus mean stock anti-hog cholera serum and stock hog cholera virus as used in the field to prevent the disease.

The Follow-Up Method

The following litters were treated by the follow-up method.

Sow No. 4

March 26 — Farrowed five pigs.
April 30 — 35 days. Each of five pigs injected with 20 cc. of serum.
May 21 — 56 days. Weaned.
May 28 — 63 days. Each of five pigs injected with 30 cc. of serum and 2 cc. of virus.
June 18 — 84 days. Moved to cholera free ground.
Nov. 26 — 8 months. Weighed and exposed to virus as shown in Table I.

Sow-Litter 6 Pig 4

May 1 — Farrowed twelve pigs.
May 2 — Three pigs taken out of litter and placed with a sow identified as Litter 5 Pig 2, which will be given later.
May 28 — 27 days. Each of nine pigs given 15 cc. of serum.
June 25 — 55 days. Each of nine pigs given 20 cc. of serum.
June 26 — 56 days. Weaned.
July 23 — 83 days. Each of nine pigs given 30 cc. of serum and 2 cc. of virus.
Aug. 5 — Pig No. 1 died as a result of an operation for hernia.
Aug. 12 — Moved to cholera free ground
January 1, 1926 — 8 months. Weighed and exposed to virus as shown in Table I.

Sow No. 5

June 13 — Farrowed ten pigs.
June 14 — Four pigs found dead.
July 3 — 20 days. Each of six pigs given 10 cc. of serum.
July 24 — 41 days. Each of six pigs given 15 cc. of serum.
Aug. 10 — 58 days. Weaned.
Aug. 22 — 70 days. Each of six pigs given 30 cc. of serum and 2 cc. of virus.
Sept. 5 — 84 days. Moved to cholera free ground.
Jan. 13, 1926 — 8 months. Weighed and exposed to virus as shown in Table I.

Table I

Group Treated by the Follow-Up Method

Identification of Litters	Average weight of litters, lbs.	Number in litters	Virus injected at 8 months cc.	Results of exposure to virus
No. 4	206.2	3	2	Remained well.
		2	10	Remained well.
*L6 P4	161.1	4	2	Remained well.
		4	10	Remained well.
No. 5	205.3	3	2	Remained well.
		3	10	Remained well.
Total	.....	19	.....	

* L — Litter; P — Pig.

No symptoms characteristic of cholera appeared in this group as a result of the exposure to virus.

THE SIMULTANEOUS METHOD

Pigs in the following litters were treated when three weeks of age by the simultaneous method.

Sow No. 2

April 29 — Farrowed twelve pigs (eight alive and four dead).
May 2 — Pig found dead.
May 20 — 21 days. Nos. 1, 2 and 3 each given 10 cc. of serum and 1 cc. of virus. Nos. 4, 5 and 6 each given 20 cc. of serum and 2 cc. of virus. No. 7, decidedly the runt of the litter, given 2 cc. of the virus alone.

The doses of serum and virus given Nos. 1, 2 and 3 were doubled for Nos. 4, 5 and 6 in order to observe whether the physical development of the pigs and the grade of immunity would be influenced by the size of dose used.

The No. 7 pig was given virus alone, as has been the practice in previous years, to observe whether virus alone can be given with safety to pigs of this age when they are farrowed by, and are suckling, immune mothers.

May 27 — No. 7 droopy, showing clinical symptoms of cholera.
June 3 — No. 7 died of cholera.
June 24 — 56 days. Weaned.
July 15 — 77 days. Moved to cholera free ground.
Dec. 29 — 8 months. Weighed and exposed to virus as shown in Table II.

The total weights of Nos. 4, 5 and 6 that received the larger doses of serum and virus when vaccinated was thirteen pounds more than Nos. 1, 2, and 3 that received the smaller doses.

Sow-Litter 1 Pig 3

May 14 — Farrowed twelve pigs.
May 16 — One pig dead.
May 18 — Two more pigs dead.
May 19 — Another pig dead, leaving eight pigs in the litter.
June 2 — 19 days. The eight pigs of this litter were so weak and unthrifty and so severely exposed to cholera that they were each given 15 cc. of serum alone instead of waiting two more days to give the simultaneous treatment at exactly three weeks of age.
June 12 — 29 days old and 10 days after the serum alone treatment. The health of the litter had somewhat improved and the simultaneous treatment was administered. Nos. 1, 2, 3 and 4 each given 10 cc. of serum and 1 cc. of virus. Nos. 5, 6, 7 and 8 each given 20 cc. of serum and 2 cc. of virus.

July 9 — 56 days. Weaned.
July 15 — A susceptible pig was placed in the pen with this litter to check on the presence of cholera contamination.
July 21 — No. 1 pig not eating. Temperature 106.8.
July 22 — No. 1 pig's temperature 105.0 and still not eating. Moved Nos. 1, 2, 3 and 4 into a separate pen in order to give them all more room and a better chance to grow stronger.
July 23 — No. 1's temperature 104.8. Seems somewhat better.
July 30 — No. 1 pig not eating. Temperature 105.0. Seems worse.
Aug. 6 — 84 days. No. 1 pig recovered sufficiently to be castrated with other boar pigs of the litter. No. 7 had a unilateral scrotal hernia. A successful operation was done. Concluded that No. 1's trouble was not cholera because the clinical symptoms other than temperature were not characteristic of the disease, also because of the nature of the recovery. Furthermore, the susceptible pig kept with this litter did not become sick but later succumbed when it was artificially exposed to virus. The case of No. 1 was the most pronounced one of a generally unthrifty litter.
Jan. 14, 1926 — 8 months. Exposed to virus as shown in Table II.

The total weight of Nos. 5, 6, 7 and 8 that received the larger doses of serum and virus when vaccinated was 95 pounds more than Nos. 1, 2, 3 and 4, receiving the smaller doses of serum and virus.

Sow-Litter 4 Pig 1
May 20 — Farrowed thirteen pigs (seven alive, six dead).
June 2 — 13 days. Litter severely exposed to cholera and slightly droopy. Each of seven pigs given 15 cc. of serum.
June 12 — 10 days after the serum alone treatment, and at 23 days of age, gave each of seven pigs 1 cc of virus alone.

By thus treating the litter an attempt was made to strengthen the passive immunity in the emergency of exposure by giving the serum alone and to still keep the date of active immunization near the twenty-one day age by administering the virus alone.

July 15 — 56 days. Weaned.
Aug. 12 — 84 days. Moved to cholera free ground.
Jan. 20, 1926 — 8 months. Weighed and exposed to virus as shown in Table II.

Sow No. 3
June 19 — Farrowed fourteen pigs. All alive but small and weak. Sow large and clumsy.

June 20 — Six pigs dead.
June 22 — Two more pigs dead.
July 2 — Another pig dead; crushed by sow.
July 10 — 21 days. Five pigs of this litter had diarrhea and were not in good condition to be treated but wished to adhere to the fixed time of treatment, three weeks.

Nos. 1 and 2 each injected with 10 cc. of serum and 1 cc. of virus. Nos. 3, 4, 5 each injected with 20 cc. of serum and 2 cc. of virus.

Aug. 14 — 56 days. Weaned.
Sept. 11 — 84 days. Moved to cholera free ground.
Feb. 19, 1926 — 8 months. Weighed and exposed to virus as shown in Table II.

When eight months of age the average weight of Nos. 3, 4 and 5, that received the larger doses of serum and virus when vaccinated, was 190 pounds, 12 pounds more than the average weight of Nos. 1 and 2, that received the smaller doses.

TABLE II

GROUP TREATED BY THE SIMULTANEOUS METHOD

IDENTIFICATION OF LITTERS	Average weight of litters, lbs.	Number in litter	Virus injected at 8 months cc.	Results of exposure to virus
No. 2	185.8	3	2	Remained well
		3	10	Remained well
L1 P3	146.8	4	2	Remained well
		4	10	Remained well
L4 P1	175.2	3	2	Remained well
		4	10	Remained well
No. 3	185.2	3	2	Remained well
		2	10	Remained well
Total	.....	26	.....	

In the four litters treated at an early age with the simultaneous method, none of the pigs showed symptoms of cholera after the exposure to virus, regardless of the quantity of serum and virus used in vaccinating. A difference in grade of immunity might have been observed if larger doses of virus had been used in the exposure at eight months. The animals that received the larger doses when vaccinated consistently weighed more than the ones receiving the smaller doses, but this point must be observed on larger numbers before it can be decided what influence dosage has, if any.

The virus alone pig has in some of the previous experiments sickened after the injection, as was the case in Litter 2. This was objectionable because it meant the presence of a sick pig and a spreader of the disease in a litter simultaneously treated. Just

what influence this may have is not known, but in order to eliminate the possibility of an influence the procedure was discontinued. The virus alone method was used on entire litters and furnished a source of information on this method of vaccination.

THE VIRUS ALONE METHOD

The following litters were treated with virus alone.

Sow-Litter 5 Pig 2

April 29 — Farrowed ten pigs, four alive, five dead and one dying when found.

May 2 — Three pigs twenty-four hours old taken from Litter 6 Pig 4 sow and placed with the four pigs of this litter. The three pigs transferred will be spoken of as "the adopted pigs" while those farrowed by Litter 5 Pig 2 sow will be called "the sow's own pigs." This identification was kept until the immediate results of vaccination were observed.

May 20 — 21 days. Each of the seven pigs was injected with 1 cc. of virus alone.

May 27 — 7 days after the injection of virus. Two pigs sick.

May 30 — One of sow's own pigs dead. Diagnosis, cholera and pneumonia. Diagnosis of cholera was confirmed by injecting 3 cc. of heart's blood into a susceptible pig which succumbed to the disease.

June 1 — Another one of the sow's own pigs dead. Diagnosis, cholera.

June 4 — One of the adopted pigs dead. Diagnosis, cholera. This left four individuals in the litter: two of the sow's own pigs and two of the adopted pigs.

June 24 — 56 days. Weaned.

July 15 — 77 days. Moved to cholera free ground.

Dec. 29 — 8 months. Weighed and exposed to virus as shown in Table III.

In this litter of seven pigs vaccinated with the virus alone, three or 42.86 per cent died of vaccination. The four that withstood the vaccination also withstood the exposure to virus at eight months of age.

Sow-Litter 1 Pig 4

May 1 — Farrowed twelve pigs, three dead.

May 2 — Three pigs dead. Thirty hours after birth took the six remaining pigs from this sow, a cholera immune, and placed them with a susceptible sow that had also farrowed on May 1. The five pigs farrowed by the susceptible sow were placed on the immune sow. The trading of pigs was done in order to study further the nature of the immunity of young pigs farrowed by, and suckling, immune mothers.

Reynolds[4] reported in 1910 that young pigs from immune mothers were found to have a high grade of immunity while those from susceptible sows were found to have practically no resistance to cholera. The same investigator proved that the immunity in young pigs from immune mothers could be made permanent by administering virus alone. Regarding the cause for the presence of immunity in young pigs from immune mothers, Reynolds[5] observed in 1911 that the work as far as he had carried it would indicate that nursing had little to do with the pig's immunity, but he made no study or distinction regarding colostrum. This phenomenon is important because it is so closely linked with the immunization of young pigs by artificial means and in these two litters a study in a small way was begun.

With the exception of a slight indifference on the part of the adopted mothers and a temporary shyness on the part of the adopted pigs immediately after the exchange, no difficulties were encountered in the trade and it is doubtful whether the pigs in either case missed as much as one nursing. The pigs from the susceptible sow were somewhat stronger and more vigorous at thirty hours of age than were the six pure bred Chester Whites farrowed by the immune sow. The susceptible sow seemed upon further observation to be the better suckler and better mother.

The immune sow was kept in a pen where some cholera contamination may have existed while the susceptible sow farrowed and was kept for a time on cholera free ground. The pigs from the immune sow were given a foot and leg bath with 0.5 per cent lysol solution before being transferred.

May 23 — 22 days after birth and 21 days after the exchange. Pigs Nos. 1, 2 and 3 were given 1 cc. of virus. Nos. 4 and 5 were kept as controls. The virus alone was given to Nos. 1, 2 and 3 to learn whether in the three weeks of nursing the immune sow they had received a passive immunity sufficiently high to protect them against the injection of the 1 cc. of virus.

May 27 — No. 1 pig slightly droopy.

May 28 — No change.

May 29 — 6 days after the virus alone injection. The litter had the appearance of a serum test with Nos. 1, 2 and 3 lying sick and hidden in the litter and Nos. 4 and 5 vigorous and active.

May 30–31 — No change noticeable.

June 1 — Nos. 1, 2 and 3 gradually growing worse with cholera. Nos. 4 and 5 slightly droopy, evidently having contracted the disease by contact.

June 2—10 days after the virus alone injection. No. 3 pig dead. Diagnosis, cholera.

June 4 — (A. M.) No. 2 pig dead. Diagnosis, cholera.
(P. M.) No. 5 pig dead. Diagnosis, cholera.

June 5—No. 4 pig dead. Diagnosis, cholera.

It is interesting that the two control pigs did not show sickness as early but died almost as soon as the pigs that received the virus.
June 9—No. 1 pig so nearly dead that it was killed. Diagnosis, cholera.

Therefore, from May 23, the time when Nos. 1, 2 and 3 were artificially exposed to virus, to June 9 (17 days) the entire litter of five which had been taken from the susceptible sow and placed on the immune sow when they were thirty hours old were destroyed by cholera.

It was evident here that a degree of immunity sufficient to protect against 1 cc. of virus injected subcutaneously had not been acquired by nursing an immune sow for the period from thirty hours to three weeks of age.

Susceptible Berkshire Grade Sow.

May 1—Farrowed seven pigs, five alive and two dead.

May 2—30 hours after farrowing. Five pigs were exchanged for the six pigs of the same age farrowed by the pure bred Chester White immune sow identified as Litter 1 Pig 4.

May 23—22 days after birth and 21 days after the exchange. Pigs Nos. 1, 2, 3 and 4 were each given 1 cc. of virus. Nos. 5 and 6 not injected, left as controls. Daily temperatures were taken of the sow and no symptoms of cholera noticed.

May 27, 28, 29, 30, 31 — Nos. 1, 2, 3 and 4 were noticed to have a somewhat roughened coat and to show slight droopiness which indicated a reaction from the injection of virus, but from June 1 on, this condition rapidly disappeared.

June 13—The six adopted pigs were forty-three days old. Weaned.

June 27—In order to prove the sow's susceptibility, 2 cc. of virus was administered by deep injection into the side of the neck. Temperatures were taken as follows:

June				July				
27	28	29	30	1	2	3	4	5
2.2	3.2	3.4	6.0	6.6	7.4	6.8	5.0	Dead

July 5 — Susceptible Berkshire grade sow dead. Diagnosis, acute hog cholera.

This showed beyond a doubt that the sow was very susceptible to cholera, a fact upon which the value of this side experiment rested. It is an interesting incident that the virus was administered to her adopted pigs while suckling her without causing her to contract the disease.

It might appear that the pre-natal influence of the immune sow was the big factor responsible for the phenomenon of immunity against cholera in her young pigs. However, the fact that each of the two litters involved in the side experiment suckled their own

mothers for thirty hours must not be lost sight of and in the light of present knowledge a conclusion of the kind just mentioned might easily be erroneous.

Certain facts regarding the transmission of antibodies from mother to young are known in cattle[6], goats[7], guinea pigs[8], and man[9]. The important question concerning this phenomenon is whether the antibodies from an actively immune mother are transmitted to the offspring through the placenta or by their presence in the colostrum and perhaps later in the milk. The possibility of the milk containing immune agents has been practically eliminated by experimentation and therefore limits the question to the placenta and the colostrum. In the more recent studies the placental route seems the important one in certain species, as in man, rabbits, guinea pigs and mice; whereas in cows, goats and sheep the colostrum contains the antibodies closely related to the presence of globulins, especially euglobulins. The conference of a passive immunity to the young in the case of the colostrum seems to depend on a permeability of the digestive tract immediately after birth (absent later) which permits the antibodies to pass directly into the blood.

The writer has been unable to find where this question has been studied in swine with the antibodies of cholera or any other disease. Therefore all that can be said here is to caution against a conclusion that the transmission of the immunity seen in the litter of six pigs farrowed by the immune sow occurred in utero for, from what we know of the rapid immunizing properties of colostrum containing antibodies, it must be considered as a reasonable possibility until experiments can be performed where the exchange of pigs between susceptible and immune sows can be made before the pigs have suckled their natural mother.

Another fact that must not be lost sight of is that the decisiveness of this experiment could not be expected regularly for, as was the case in Litter 5 Pig 2, where pigs were farrowed by an immune sow and suckled an immune sow, the litter withstood the virus alone injection poorly at three weeks, three out of the seven dying from the effects of the treatment. It is therefore somewhat remarkable that the four pigs from the immune sow, suckling a susceptible sow when injected with virus alone at three weeks, lived.

Further account of the six pigs that survived the side experiment just described, follows:

Sept. 24 — No. 1 pig died after showing a low condition of health, coughing and poor appetite for two weeks. Autopsy revealed the presence of lung worms, also a quantity of gravel in the stomach and large intestines, the latter indicating a perverted appetite. Neither clinical symptoms nor post-mortem lesions were characteristic of cholera.

Oct. 16 — 138 days. Nos. 5 and 6, which had been kept as controls, were exposed to virus and found to be susceptible.

Jan. 1, 1926 — 8 months. Nos. 2, 3 and 4 were weighed and exposed to virus as shown in Table III.

Table III

Group Treated by the Virus Alone Method

Identification of Litters	Average weight of litters, lbs.	Number in litters	Virus injected at 8 months cc.	Results of exposure to virus
L5 P2	168	3	2	Remained well.
		1	10	Remained well.
L1 P4	148	1	2	Remained well.
		2	10	Remained well.
Total		7		

General Discussion

The three methods of vaccination were administered to nine litters in rotation, the follow-up method to the first litter farrowed in the spring, the simultaneous methods to the second, the virus alone method to the third, the follow-up method to the fourth, etc. Nineteen animals treated by the first method were exposed to virus at eight months of age, twenty-six by the second, and seven by the third. The greater number treated by the simultaneous method was due to the emergency treatment given to the seven pigs belonging to the sow identified as Litter 4 Pig 1. Had this litter not been so severely exposed they would have been used in the virus alone group.

The average weight at eight months of age for the nineteen animals vaccinated by the follow-up treatment was 186 pounds; of the twenty-six vaccinated by the simultaneous method, 170 pounds; and of the seven vaccinated by the virus alone method, 159 pounds.

The grade of immunity as measured in each of the three groups by the 2 cc. and 10 cc. doses of virus given at eight months of age seemed the same and requires no discussion. In the second question of the problem, regarding thrift of the groups vaccinated by the different methods, the same uniformity was not observed. There were certain factors aside from method of vaccination which seemed to have an influence on daily gains that are worthy of mention.

The arbitrary manner of choosing the time for treatment in the follow-up method as compared to fixing a definite time for treatment in the simultaneous and virus alone methods was a probable influence.

The breeding of the animals used was very nearly the same for

all three groups, but where a small number of sows is used as in this experiment, the law of chance does not often equalize and a large influence on thrift depends on the sows that happen to fall in a group. The differences in maternal characteristics are hard to show in data. The weights in the various groups are a good index of maternal influence as observed in this experiment.

Environment after weaning also plays a part. All pigs, except those from No. 5 and No. 3 sows, were kept together through the summer until October 16 when quarters became crowded and seven from Litter 1 Pig 3, two from Litter 6 Pig 4 and three from Litter 4 Pig 1, were moved to a separate pen to relieve congestion and receive closer attention since they were some of the poorest animals in the herd. Litters from Nos. 3 and 5 were kept together after being moved to cholera free ground and were never placed with the remainder of the pigs in the experiment. The care of these two litters was practically the same from time of vaccination until time of their exposure to virus. Their chances at feed and shelter were more nearly equal than was the case in the larger herd which consisted of the remainder of the litters.

SUMMARY

1. All animals withstood the 2 cc. and 10 cc. doses of virus alone administered at eight months of age, regardless of method of vaccination used and regardless of quantities of serum and virus used in each treatment.

2. The animals vaccinated by the follow-up method weighed more than those vaccinated by the simultaneous method, and those vaccinated by the simultaneous method weighed more than those vaccinated by the virus alone method. Also, the animals receiving larger doses of serum and virus at three weeks of age weighed more than the ones receiving the smaller doses at that age. However, this relationship between weights and methods of vaccination is far from being established by this one experiment. There are numerous factors, some of which have been pointed out in the general discussion, that must be considered before a conclusion can be drawn. A very large number of experiments will be needed *if the litter is kept as the unit of comparison,* for the mean of natural individual differences of litters must be established by using animals of the same breeding, kept under the same conditions with regard to feeds and feeding, housing and other phases of management. The number of fetuses expelled, which frequently has a bearing on their size and strength, the number that survive, the good suckler and careful mother, as compared to one poor in these characteristics, are other factors of special significance when comparing the weights of few litters.

3. In summarizing the one study on the phenomenon of the presence of immunity in young pigs farrowed by an immune mother, it can be said with reasonable sureness that six pigs farrowed by an immune sow and nursing her for thirty hours re-

ceived, either in utero or from her colostrum, a grade of immunity sufficiently high to protect the four of them against 1 cc. of virus after suckling for three weeks a sow that was later proven to be very susceptible to cholera. The five farrowed by the susceptible sow and nursing her thirty hours contracted cholera and died in a way typical of the disease, three by artificial injection and two by contact, after they had suckled the immune sow for three weeks.

References

1. Benner, J. W. Report of the New York State Veterinary College, 1922–1923, p. 204.
2. Benner, J. W. Report of the New York State Veterinary College, 1923–1924, p. 87.
3. Benner, J. W. Report of the New York State Veterinary College, 1924–1925, p. 225.
4. Reynolds, M. H. Am. Vet. Rev., xxxviii (1910), p. 236.
5. Reynolds, M H. Am. Vet. Rev., xl (1911), p. 485.
6. Smith, T. The Cornell Veterinarian, April, 1925, p. 173.
7. Famulener, L. W. J. Inf. Dis., 1912, x, p. 332.
8. Hagan, W. A. J. of Exp. Med., Dec., 1922, xxxvi, No. 6, p. 697.
9. Kuttner, Ann, and Ratner, Bret. Am. J. Dis. Children, 1923, xxv, p. 413.

THE PASSAGE OF BACTERIUM ABORTUM THROUGH THE DIGESTIVE TRACT OF CATTLE

HERBERT L. GILMAN AND R. R. BIRCH
Veterinary Experiment Station

In the study of any infectious disease, two of the most important factors are the methods of elimination of the causal agent from the body, and its method of transmission to susceptible animals. In Bang abortion disease, the uterine discharge is without doubt the most important method of elimination of the organism (*Bact. abortum*), and it has been quite conclusively proved that ingestion with the food is an important source of infection of other animals. The part played by the milk in the spread of the disease is not so clearly understood. Other means by which bacteria are taken into the body, such as through the conjunctiva, teat canal, etc., have not received extensive study, and are probably of less frequent occurrence. If we are to control the disease entirely, however, we must take into consideration all the possible methods of elimination and infection.

While the dissemination of the organism through the feces is probably a minor factor in the spread of the disease, we believe it should receive due consideration in our study of the problem. The presence of this organism in the feces is important not only because of its possible role in the spread of the disease, but also because of the possibility of the contaminated feces soiling the vagina and thus infecting the animal through this channel rather than through the digestive tract. In a previous paper we stated: "Investigators have quite generally assumed that evidence of infections following oral administration of cultures was necessarily brought about by the absorption of the organisms through the mucosa of the digestive canal and their subsequent distribution through the general agencies of the lymph and blood streams to the pregnant uterus and other parts. Years ago Williams pointed out the fallacy of this assumption, calling attention to the fact that the organisms may pass unharmed through the digestive canal, and thus reach the vulva which often becomes soiled with feces." The passage of the organisms unharmed through the digestive tract is therefore important for more than one reason.

In the paper cited above we demonstrated that the bacteria could pass unharmed through the digestive tract, though the number of organisms ingested was rather large, and the number of positive findings small. The dose given the cow was one liter of a twenty-four hour bouillon culture of *Bact. abortum*. Three of the twenty-four guinea pigs injected with extract made from the feces discharged following oral administration of the culture were infected with the organism. The other died too soon for lesions to develop, or showed no evidence of infection.

Barger and Hayes experimented with this problem using six young calves under seventy-two hours of age. These were placed in three groups of two each. Group "A" was fed artificially infected milk. Twelve of the fourteen pigs inoculated with feces, taken after administration of the infected milk, became infected with *Bact. abortum.* Group "B" was also given artificially infected milk. Most of the pigs inoculated with the feces of calves in this group died during a general epidemic among their pigs, but nevertheless two out of the twenty were positive. In group "C," one calf was fed artificially infected milk, and the other milk from a cow known to be discharging the organism from an infected udder. Four out of twelve of the pigs injected with feces from the calf fed artificially infected milk showed positive evidence of infection, and six out of twelve injected with feces from the calf fed the naturally infected milk were positive. The authors conclude: "1. *Bact. abortum* passed through the entire digestive tract of suckling calves fed milk either artificially or naturally infected. "2. The organisms retained their virulence for guinea pigs after passage through the digestive tract of the calf."

During the course of an experiment conducted recently in order to study the effect of ingestion by pregnant cows of suspensions of live cultures of *Bact. abortum,* we decided to repeat our previous experiment using a larger number of animals. Eight pregnant heifers were used in the test. Each animal received three times weekly ten cubic centimeters of a saline suspension of a twenty-four hour agar growth of *Bact. abortum.* The density corresponded to a reading of a .75 on the Gates scale. This dose was much smaller than that used in our previous experiments. The suspension was diluted with physiological saline solution and sprinkled over the feed. Samples of feces were collected from each cow on five occasions, and the guinea pigs injected were divided into groups A, B, C, D, and E. The technique of collecting the feces was the same as that used in our previous experiment. The method of preparing samples for the guinea pig inoculations in Group "A" was as follows: About 200 grams of the feces were mixed with about 200 cc. of physiological saline solution, and filtered through several thicknesses of cotton gauze. Because of the high mortality in this group, the suspension injected into the other groups was diluted with an equal quantity of 1–1000 gentian violet solution and allowed to stand in the refrigerator over night. The gentian violet was used because of its inhibitory action on the growth of contaminating Gram positive bacteria. One cubic centimeter of feces suspension was injected subcutaneously into each guinea pig. In five weeks the pigs were killed and examined for evidence of infection with *Bact. abortum.*

The results are summarized in the following table:

GROUP	Samples collected	Last feeding prior to taking feces sample	Interval between last feeding and collection of sample	Number of pigs injected	Number of pigs died of sepsis	Number of pigs autopsied	
						Neg. to Bact. abortum	Pos. to Bact. abortum
	1925	1925					
A	Sept. 15	Sept. 12	3 days	19	16	2	1
B	Sept. 22	Sept. 19	3 days	16	6	8	2
C	Sept. 29	Sept. 26	3 days	16	9	4	3
D	Oct. 5	Oct. 3	2 days	14	8	6	0
E	Oct. 12	Oct. 10	2 days	16	7	9	0
				81	46	29	6

Summing up the results, we find that of the eighty-one pigs injected, forty-six died of sepsis. Of the thirty-five that came to autopsy, six were positive to *Bact. abortum,* and twenty-nine were negative. The mortality rate was rather high, but this was to be expected. Perhaps this might have been overcome to some extent had we passed the feces suspension through filter paper, as was done by Barger and Hayes, rather than through gauze. The work shows, however, that *Bact. abortum* does, at times, pass through the entire digestive tract and retain its pathogenicity for guinea pigs. While the number of positive pigs was not high, it is well to consider that the dose of organisms given the cattle was comparatively small (10 cc. of a suspension of a density .75 on the Gates scale). The relatively higher percentage of positive findings observed by Barger and Hayes might have been made possible by the milk diet in the calves favoring viability of the organisms. It will be observed that all of the pigs which were positive to *Bact. abortum* were injected with feces samples obtained three days after the last culture was fed. While the pigs injected with samples taken following a two-day interval were all negative, the number of pigs involved was not sufficient to render comparisons between the two groups of any value.

DIRECTIONS FOR COLLECTING BLOOD SAMPLES FOR THE AGGLUTINATION TEST FOR INFECTIOUS ABORTION

C. M. CARPENTER

Diagnostic Laboratory—Department of Pathology and Bacteriology

As our knowledge concerning infectious abortion due to *Br. abortus* increases, we are beginning to realize the value of the agglutination test as an aid in controlling this widespread disease. Although errors in experimental work cannot be entirely eliminated and difficulty in interpreting certain reactions is experienced, we believe that the test is of great aid in detecting infected animals.

Of necessity this test must be made in a laboratory and by veterinarians or other workers who have had experience with serological examinations. In order to obtain the best results in this work it is necessary to have a good sample of blood from each animal. Only the serum from the blood sample is used and it is obvious that enough blood should be drawn to give a sufficient amount of serum to make the test after the blood has clotted.

Because the laboratory has in the past received blood for this test in all types of containers, the majority being very poor, we have decided that much of our time can be saved by furnishing veterinarians with sterile containers of the proper type so that we are insured of securing good samples of blood serum. *Upon receipt of a request stating the number of bottles required, they will be forwarded promptly. A charge of twenty cents per sample is made to cover the cost of making the test and of the container.* Heretofore much time has been consumed by breaking up the samples of clotted blood received in unsatisfactory bottles and by centrifugation. When large numbers of samples are to be tested, the less the individual sample is handled the better it is for the laboratory as well as for the individual for whom the test is being made.

RESTRAINT OF ANIMALS

Wherever possible the bleeding should be carried out in the stable with the animal secured in a stanchion, although if an animal is in a box stall it can be bled as a rule without difficulty unless it is a vicious bull. It is almost a necessity to have an attendant secure the animal's head fast. This is best done by means of nose tongs (cattle leader). If a stable is not available and it is necessary to collect the samples in a pasture, the animals may be corralled in a chute or a corner of the fence. If one is unable to get to the animal's head he may, with a sharp scalpel, cut the ventral coccygeal artery of the tail a short distance from its base and usually obtain a large enough sample. I have seen this method used in many instances without apparent detriment to the animal. Placing the thumb and forefinger in the animal's nostrils and pinching tightly will serve as enough restraint with some groups of animals.

PROCEDURE

After the nose tongs have been placed, the head of the cow should be elevated and turned to the side opposite which the operator prefers to work. This extends the neck so that the jugular groove is easily visible and the skin over the vein more or less stretched. An antiseptic is placed on the skin over the vein where the needle is to be inserted. The jugular vein is compressed with the left hand, the vein being distended and easily observable if the head is in the proper position. Occasionally one will find it necessary to use a choke rope to distend the jugular vein, but as a rule a sample of blood can be obtained in about the same length of time that is required to place the rope in position. With the right hand the needle (See "Suggestions Concerning Needles") is pushed through the skin at a point about six inches posterior to the angle of the jaw and with a quick thrust inserted into the vein. When the needle is in the vein a good stream of blood flows. If only a few drops of blood come from the needle, the chances are that the needle has gone through both walls of the vein and the blood is coming from the tissue outside the vein into which the hemorrhage has occurred. Retracting the needle slightly will often bring it back into the lumen of the vein. Much time may be saved by having a second attendant hand the operator the bottles and do the labeling.

SUGGESTIONS CONCERNING NEEDLES

Each veterinarian has some particular type of hypodermic needle that he prefers to use for bleeding cows. Our experience has shown that a needle with a comparatively large gauge is most satisfactory for two reasons. The needle with a small lumen becomes plugged easily because the blood clots more quickly. The larger-gauged needles fill the bottle more rapidly and with nervous animals it saves trouble if the sample is obtained in the shortest period of time. An animal will often jerk away suddenly and a second puncture of the vein is required if a needle is used that will allow only a small stream of blood. The needle ought not to be too short. *We prefer a* 12 *or* 14 *gauge needle from one and one-half to two inches long. By all means use sharp needles. It is not necessary to use a separate needle on each animal.* The needle should be surgically clean. It is our practice to push a stylet through the needle after a sample has been drawn to free it from any particles of clotted blood. It is then rinsed in warm water to which may be added a little disinfectant. The rinsing of the needle may be omitted but it is advisable.

CONTAINERS, LABELING, SHIPPING

The serum from a bovine blood clot does not separate as readily as does that from the horse or other species of animals. We have observed that the blood clot squeezes out serum well in some containers while in others no separation occurs. *We have found that a ½-ounce oval bottle is by far the most satisfactory under all con-*

ditions. A rubber stopper or cork of good quality should be used. The bottle should be filled only half full of blood. (See plate.) It is important to have the blood drawn into a sterile bottle if possible, and to have the bottle dry before the sample is taken. If the blood becomes badly contaminated, bacteria will multiply in transit if the sample becomes warm, because blood is a very favorable medium for their growth. The bacteria destroy the blood cells and the serum becomes discolored by the hemoglobin. If the samples are drawn into clean containers and shipped immediately they usually arrive in good condition. But if any water or disinfectant is left in the bottle, hemolysis occurs because the former are not isotonic with the blood. If the serum contains much hemoglobin it interferes with the reading of the test.

Each bottle should be labeled distinctly so that no error will occur in reading the names or numbers on the samples after they reach the laboratory. The bottles should be wrapped separately in pieces of paper or separated by pieces of cardboard and *shipped via parcel post. It is better, where large numbers are packed together, to have them stamped and labeled, "Special handling." Special delivery is also advisable,* although it is not necessary. Icing of the samples is not required.

A.—One-half ounce empty bottle of correct size and shape for the blood sample.
B.—The bottle was half filled with blood. Note the separation of the serum.
C.—The bottle was completely filled with blood. Practically no serum has separated from the clot. This is an unsatisfactory sample.

THE VALUE OF PLACENTAL EXAMINATION FOR THE DIAGNOSIS OF INFECTIOUS ABORTION OF CATTLE*

WILLIAM A. HAGAN

Department of Pathology and Bacteriology

In the course of a bacteriological study of bovine placentae search of the membranes was made for the specific lesions of *Brucella abortus* infection. Later these data were correlated with the clinical history and with bacteriological evidence relating to the presence or absence of the Bang bacillus in the content of the uterus. So far as is known at present the lesions of the placenta are the only gross changes due to the abortion organism which may be identified with reasonable certainty without laboratory assistance of some kind. If proper attention is paid to these changes it is believed that the clinician can make a diagnosis of infectious abortion due to *Br. abortus* in herds under his charge without any outside assistance and with a high degree of accuracy.

Lesions of the Bovine Placenta Due to Brucella Abortus

In his first work on infectious abortion, Bang[1] found a pasty, odorless, brown-colored exudate lying between the chorion and the wall of the uterus. In this exudate he found microscopically many minute bacilli which he succeeded in cultivating and which are now known as *Brucella abortus*. Most of the bacilli were free, but some were packed in dense masses which appeared to lie within cells. Bang considered the exudate to be a product of the uterine wall rather than of the chorion and this led him to consider the process to be a chronic uterine catarrh, although the uterine mucosa showed little evidence of disease.

Theobald Smith[2] pointed out the fact that the epithelial layer of the chorion appeared to be a seat of multiplication of abortion bacilli and that the bacilli-choked cells so frequently found in uterine exudates following abortion were derived from this membrane. Since these cells are not phagocytic, the method by which the bacteria invade them is obscure but there is no doubt that, once inside, they multiply until the cells are filled with them. This infection of the chorion brings about progressive changes which may be recognized in the gross.

There does not appear to be anything in the cotyledons themselves which will indicate when *Br. abortus* infection is present. The cotyledons of the affected part are always more or less necrotic but the soft tissue of the cotyledon is subject to rapid degeneration, so if the membranes have been retained for any length of time decomposition will have progressed to the point that detection of previously existing necrotic changes is uncertain, or impossible.

* The pathological material and the clinical history of the cases described in this article were furnished by the staff of the Ambulatory Clinic. My thanks are due to Drs. D. H. Udall, M. G. Fincher and E. R. Cushing for their hearty cooperation.

Fig. 1. Portion of chorion showing advanced stage of disease caused by *Brucella abortus* infection. The cotyledons are necrotic but the most striking and characteristic lesion is the leather-like thickening and wrinkling of the inter-cotyledonous part of the membrane.

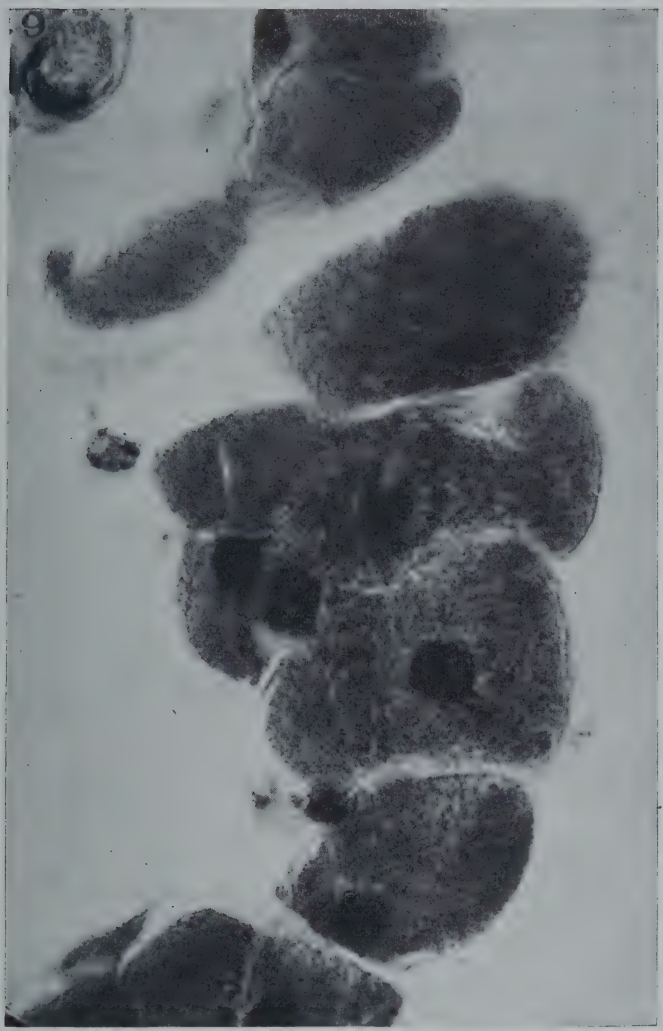


Fig. 2. Epithelial cells of the outer surface of the chorion which are heavily loaded with the Bang bacillus. The normally clear cytoplasm is in this case heavily stippled because of the bacilli. The nuclei are not invaded but usually become pyknotic. With methylene blue or other basic stains the invaded cells are conspicuous because of the depth of the stain which is retained by the bacillary masses. (After Smith, Theobald; Jour, Exp. Med.)

Furthermore, necrotic cotyledons are seen in some fresh placentae which are not infected with *Br. abortus.*

The characteristic lesions of *Br. abortus* infection are to be sought in the smooth, free, intercotyledonous chorion. This portion is not quickly altered by decomposition, so the specific lesions can be found and recognized in membranes which have been retained for several days and are exceedingly putrid. The lesion may be widespread or it may involve only a small area of the chorion. The extent of the lesions is no index to the clinical behavior of the animal. Some animals with an area only as large as a man's hand abort while others with a large part of the chorion affected either abort late or carry the calf to full term. That the lesions may be confined to a small area should be borne in mind when examining placentae. Every effort should be made to obtain all of the membranes and all parts should be examined.

The lesions invariably surround one or more cotyledons and possibly radiate from these structures. In the earliest stages, the infected intercotyledonous chorion loses its moist transparent appearance and becomes dull and dry. The blood vessels become engorged so there is reddening of the part. Later the membrane becomes more opaque, becomes thickened, wrinkled, brownish and leather-like. At the periphery of the leather-like area an active zone of inflammation is to be found. Here the chorionic surface is just becoming dull, and here one can usually find many abortion bacilli-choked cells by scraping and making microscopic preparations of the material obtained. In the older part of the lesion—the greatly thickened, leather-like portion—it is difficult, or impossible, to find organisms in smear preparations.

The diseased chorion is in most cases more or less edematous, the fluid-filled connective tissue between the epithelial layers sometimes becoming an inch or more in thickness. Edema of this membrane is sometimes seen without infection and sometimes infection occurs without the edema, hence the condition is of little diagnostic value.

In searching for lesions of *Br. abortus* in bovine placentae, one must be on his guard not to mistake the peculiar leather-like mummification which almost always is to be found at the tips of the horns. This is a condition of dry gangrene apparently due to failure of the blood supply to the part. It has nothing to do with Bang bacillus infection.

In the present study in a series of about 100 placentae which were collected in a routine manner by the Ambulatory Clinic of the New York State Veterinary College, thirty-two came from cases which the clinicians diagnosed as abortions. In some instances the membranes were expelled promptly and were thus secured in a relatively fresh condition; in others where retention of the membranes occurred, they were not secured for several days and consequently they were in a more or less putrid condition when examined. These membranes were spread out on a large table, examined carefully and a diagnosis of *Brucella abortus* infection or absence of abortus infection was made on the character of the

gross lesions alone. The diagnosis was checked subsequently by a microscopic examination and in most cases by guinea pig inoculation. In most cases an agglutination test of the blood was also made. The animal inoculations and agglutination tests were made for the most part by Dr. H. L. Gilman. Some were made by Dr. C. M. Carpenter. To them my thanks are due. The general results of the tests are contained in the Table.

TABLE I

No.	Placentae lesions	Placenta microscopically	Agglutinins for Br. abortus blood	Milk*	Uterine exudate*	Fetus*
1	+	+	1–160	..	..	..
2	?	+	1–160	—	+	—
5	+	+	1–640	+	+	..
6	+	+	.....	..	+	..
9	—	—	1–640	—	+	..
10	+	+	1–640	+	+	..
13	—	—	.....	..	..	—
14	+	+	1–60	..	..	..
15	+	+	1–320	..	+	—
16	—	—	.....	—	..	—
21	+	+	1–160	+	+	..
22	+	+	1–360	..	+	+
24	—	—	.....	—	—	—
28	—	—	1–120	..	+	—
29	+	+	1–640	—	+	..
30	+	+	1–160	+	+	+
36	+	+	1–640	..	+	+
49	—	—	.....	—	—	..
74	—	—	1–160	..	+	+
75	+	+	.....	—	—	..
84	—	—	1–80	—	—	—
87	+	+	1–640	+	..	..
92	—	—	.....	—	—	—
98	+	+	1–320	—	+	+
99	—	—	1–20	—	—	..
100	+	+	1–640	—	..	..
101	+	+	1–640	..	+	..
102	+	+	1–60	..	..	..
103	+	+	1–20	..	..	..
104	—	—	1–20	—	—	..
105	—	—	1–20	—	..	..
106	+	—	1–80	—	+	..

* Results of guinea pig inoculation.

Summarizing the data in Table I, it can be seen that all of the cases (nineteen) diagnosed as positive by the placental examination proved to be positive by one or more of the other tests. In 14 instances animal inoculation proved the cases positive, in one instance animal inoculation proved negative and in 4 instances animals were not inoculated.

The macroscopic diagnosis was confirmed by microscopic examination of the placental lesions in all cases but one. In Case No. 106, the gross diagnosis was adhered to although the organisms could not be found in smears. The lesions were very old and the organisms were no doubt very scarce. The correctness of the diagnosis was confirmed later by guinea pig inoculation. In Case No. 2, the lesions were in a very early state of development and had not developed their characteristic appearance. There was some doubt, therefore, as to their nature until a microscopic examination had been made. The organisms were demonstrated easily in this instance.

Of the 12 cases diagnosed as negative by placental examination, 9 proved negative by all other tests which were applied. In three cases, however, animal inoculation showed the presence of *Br. abortus*. In these instances, therefore, a correct diagnosis was not arrived at by placental examination alone. In one of these three cases (No. 74) only a fragment of the non-gravid chorion containing but a single cotyledon was submitted for examination—most of the membranes were not seen. In the second instance (No. 28) the membranes were mummified and enclosed a small mummified fetus—the mummifying process successfully obscuring all lesions which may have been present. In the third instance (No. 9) the membranes were in reasonably good condition and the lesions were probably overlooked. On the other hand we have in case No. 75 an instance in which the placental examination was positive, macroscopically and microscopically, while the other methods of diagnosis, including guinea pig inoculation, failed.

In this series of 32 clinical abortions, all available evidence points to the fact that 23 were due to *Br. abortus* and 9 were due to other causes. On this basis, a correct diagnosis was given in 29 instances and incorrect in only three. This represents an accuracy of about 90 per cent, a surprising degree of accuracy for such a simple test.

Summary:

Gross lesions of characteristic appearance are produced in the placenta through the workings of the *Brucella abortus* Bang. These represent the only gross lesions due to this organism which may be recognized positively in the bovine species. The lesions are located in the chorion and are particularly characteristic in the portions of this membrane which lie between the cotyledons. The lesions may be widespread or may occur only in a circumscribed area. When abortion is due to *Brucella abortus* it is believed that these lesions are always present and may be recognized in the majority of cases. In the present instance, a correct diagnosis was made in 29 cases in a series of 32 abortions.

BIBLIOGRAPHY

BANG, B. Jour. Comp. Path. and Therap., 1897, **10,** 125.
SMITH, THEOBALD. Jour. Exp. Med., 1919, **24,** 451.

BACILLARY WHITE DIARRHEA

E. L. BRUNETT
Department of Pathology and Bacteriology

The hatching of eggs from February until June is the seasonal work on the majority of poultry farms in New York State. During this period, preparations are carefully made for the obtaining of eggs of desirable stock which will give chicks of superior quality. The short maturing period and life of the fowl has added very much to the success attained in improving the different breeds. The poultryman can improve his stock to a greater extent in a few years than can the breeder of our large domesticated animals. Unfortunately, consideration for the different latent diseases which may affect poultry has not been seriously considered in the breeding program. Hence, the dissemination of certain maladies by means of valuable breeding fowls has arisen. When a desirable type of stock has been obtained as the result of careful selection and breeding, it is indeed disrupting to later introduce a disease by the addition of breeders from outside.

Inasmuch as some poultrymen do not find it practical to hatch their own eggs, the baby-chick business has reached enormous proportions. This business may be divided roughly into two groups, one being those farms where eggs from their own flocks are hatched, and the other group comprising the large hatcheries where all of the hatching eggs are purchased from neighboring flocks. In this latter group the control of the quality of the eggs is not as readily accomplished as in the former. For this reason the incidence of diseases among the chicks is expected to be higher.

One of the most common diseases of chicks that the hatcheries and poultrymen have to contend with is bacillary white diarrhea.

Occurrence. This disease is prevalent among the flocks of this state and numerous other states as well. During the hatching season it stands foremost as one of the important diseases. Extensive measures have been pursued in other states to control it and establish flocks free from this disease. This has been especially true of flocks where a particular breed has reached a high perfection of development.

Etiology. Bacterium pullorum, the causative organism, is a non-spore forming rod belonging to the paratyphoid group of bacteria. It is non-motile and does not retain the Gram stain.

On beef-infusion agar it develops discrete translucent colonies, 0.5 to 1 mm. in diameter, which have the appearance of dew drops. This clear crystal appearance of the colonies permits them to be differentiated from the colon bacillus and other contaminating types encountered in culturing carcasses. The organism grows best aerobically at 37.5° C. Twenty-four hours of incubation are usually sufficient for the development of recognizable colonies. Occasionally cultures made from infected organs will not show an appreciable growth until after forty-eight hours of incubation.

The majority of strains are aerogenic, or gas producers, but occasionally anaerogenic types, or non-gas producers, are encountered. The importance of the medium used for the determination of this property of bacteria has been shown by a number of workers. In this laboratory practically all of the pullorum strains isolated during one hatching season from specimens forwarded from outbreaks were of the anaerogenic type. Beef-extract bouillon was being used as the basic medium. Gas-producing types of other bacteria did not appear to have this property altered. The following year beef-infusion bouillon (sugar free) was used as the basis for the sugar tests. Practically all strains isolated were of the aerogenic type. *Bacterium pullorum* ferments dextrose and mannite; and fails to ferment lactose, sucrose and maltose. These sugars, being commonly used in routine laboratory work, enable sufficient information to be obtained so that a diagnosis can readily be made.

The organism is readily destroyed by disinfectants, as shown by Jones(1). Corrosive sublimate 1: 1000 kills in 30 seconds; 1 per cent carbolic acid kills in 5 minutes; 5 per cent carbolic acid kills in 30 seconds; and 1 per cent creolin kills in 5 minutes.

Forms. The disease may be classified as adult, and chick infection.

Adults, chronic form. It is in the adult that the disease may be said to exist in a latent state, the infected adult being known as a "carrier." Localizing in the ovary the infection may produce visible autopsy lesions in the form of abnormal ova. The normal ova vary in size from minute white globules to large yellow, plump spheres. The infection of the ovum may render it reddish or dark yellow in color, and irregular in shape. (Fig. 3) The mistake should not be made in calling all irregular shaped and abnormal colored ova pullorum infected. Other infecting organisms and non-bacterial causes of degeneration cannot be entirely eliminated with the naked eye.

The organisms, when localized in the ovary, are able to leave the body of the hen by means of the egg. (Fig. 1) All of the eggs laid by a "carrier" do not contain the organisms. The percentage of eggs that are infected from such hens varies with different individuals. The examinations that have been made of the eggs from infected hens show that in some cases one egg in twenty is infected, but in other cases a larger number of infected eggs were found.

The infection may not involve the ovary, but localize in other body tissues. Gross lesions of such a localization are rarely found. The importance of this type of infection does not appear to have been entirely solved. A more extensive and systematic search for *Bacterium pullorum* in other tissues besides the ovary may show that it is frequently present elsewhere.

Rettger, Kirkpatrick and Jones(2) in 1915 reported the finding of *Bacterium pullorum* in the testes of two adult male birds In 3 others it was isolated from the pericardial sac. In still another male it was obtained from both the testes and pericardial sac.

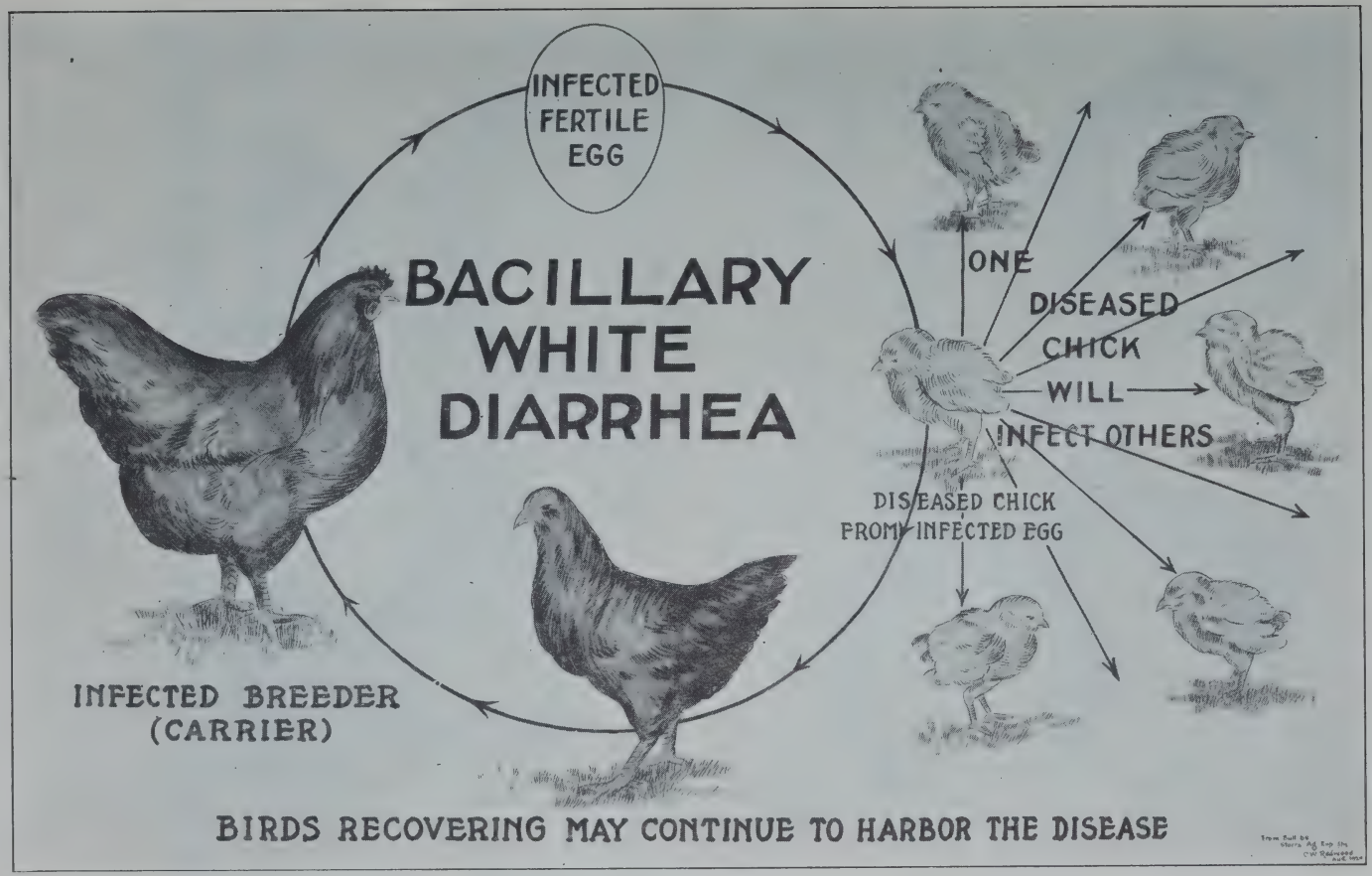
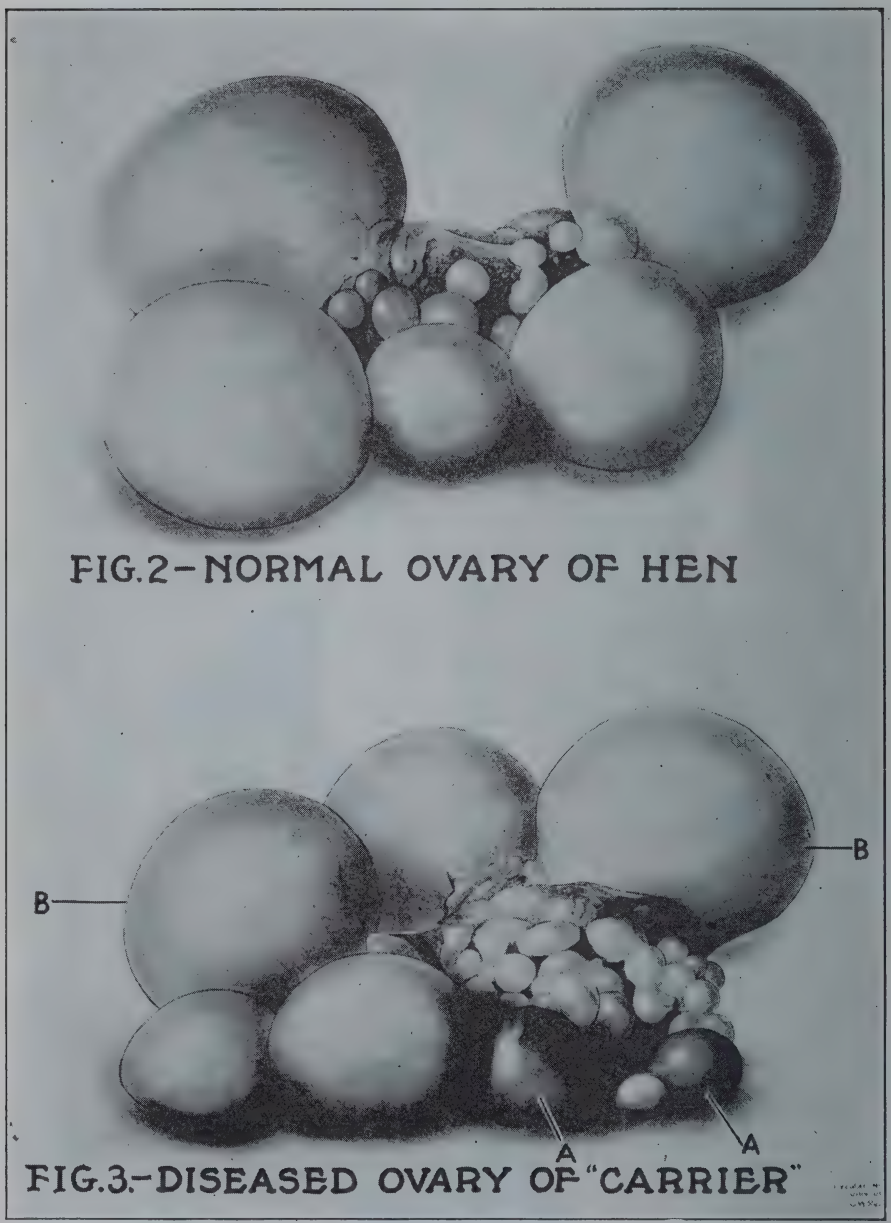

INFECTED FERTILE EGG
BACILLARY WHITE DIARRHEA
ONE DISEASED CHICK WILL INFECT OTHERS
DISEASED CHICK FROM INFECTED EGG
INFECTED BREEDER (CARRIER)
BIRDS RECOVERING MAY CONTINUE TO HARBOR THE DISEASE

Fig. 1. Cycle of infection.



A. Diseased ova.

B. Normal ova

Doyle(3) reports the finding of *Bacterium pullorum* in various tissues as follows:

Number of chickens examined	Number found infected	Number showing pathological ovaries
42	37	29

Bacterium pullorum isolated from

Ovary	Spleen	Gall bladder wall	Kidney	Liver	Bile
37	11	9	4	2	1

Jones has called attention to the fact that eggs artificially inoculated with *Bact. pullorum* did not give a high percentage of hatches. Beaudette, Bushnell and Payne (4), investigating the cause of the low percentage of hatches in a large hatchery, incriminated *Bact. pullorum.* The chief danger, however, lies in the fact that a few infected chicks may spread the infection to others in the same brooder.

Adults, acute form. Bacterium pullorum is occasionally found as the cause of acute disease among adults. Autopsy invariably reveals evidence of a septicemia. "Carriers" developing a septicemia with a fatal termination are not uncommon. The difficulty of accurately determining the source of the infection does not allow the localized infection to be incriminated in all cases. This possibility, however, cannot be lost sight of. These spontaneous cases of acute pullorum infection are thought to cause mild epizootics which appear in infected flocks.

Jones(5) has reported the occurrence of an acute disease among adults caused by *Bacterium pullorum.* Infected eggs which failed to hatch were fed to a flock with fatal results. The flock was considered free from the infection before the eggs were fed. The following year the offspring from the flock suffered from bacillary white diarrhea. This indicated that a number of survivors of the acute infection had become "carriers."

Chicks, acute form. It is in the chicks that the disease manifests itself most conspicuously. A chick affected with the acute form (Fig. 1) either leaves the egg in an infected state or becomes infected from a contaminated brooder. As shown by Jones (1), the chick is most susceptible during the first 24 hours of life.

In the following experiment the chicks were fed 0.15 cc. of a 48-hour bouillon culture of *Bact. pullorum:*

	Pos.*	Neg.†
Pen 1. 16 chicks 24 hrs. old	13	3
Pen 2. 16 chicks 48 hrs. old	5	11
Pen 3. 16 chicks 3 days old	1	15
Pen 4. 16 chicks 4 days old	0	16
Pen 5. 16 chicks 5 days old	0	16
Pen 6. 16 chicks checks	0	16

* Pos.— Chicks died. Infection proven by the isolation of the organism.
† Neg.— Chicks lived.

In another experiment the same investigator was able to infect 14 out of a total of 18 chicks by placing them in the brooder used above, 11 days after the last chick died in it. Later the brooder was disinfected and 24 chicks reared successfully in it.

Hinshaw, Upp and Moore(6) have demonstrated some interesting facts in their "Studies in Transmission of Bacillary White Diarrhea in Incubators." In this preliminary report on the bacteriology of incubators, they state that bacteria are more numerous at the time of hatching than during the period of incubation. Two fecal types were encountered. Although they were unable to isolate *Bacterium pullorum* from the incubator which was hatching infected chicks, later in an artificially infected machine isolation of *Bacterium pullorum* was readily accomplished. Half-day old chicks were killed by swabbing the nostrils and injecting one drop into the nostrils of a 24-hour culture of *Bacterium pullorum.*

They were also able to disseminate bacillary white diarrhea by placing artificially infected chick down in a forced-air-draught-type of incubator. Similar results were obtained by infecting the down of hatching chicks and exposing healthy chicks to the down which might be carried from the infected chicks to the non-infected.

Symptoms. These are not entirely pathognomonic. The infected chicks crowd near the brooder-stove or other places where extra heat is obtainable. They chirp frequently and have a hunched up appearance. The absence of general liveliness is conspicuous. Diarrhea may be present causing a matting of the down around the vent.

Autopsy. The autopsy lesions among day old chicks are not diagnostic. The history of an unusual mortality during the first few days of life would suggest some infection.

Congestion of the internal organs may be present. An unabsorbed yolk sac is invariably found. Its diagnostic importance is doubted. Films made direct from the heart blood and liver usually reveal only a small number of bacteria. Cultural methods alone must be depended upon for an accurate determination of the infection.

Chicks, chronic form. The disease in this form is apparently due to a late infection from infected chicks, contaminated brooders or an early infection with an organism of comparatively low viru-

lence. As the disease spreads among a flock of chicks the increase in age of the chicks tends to transform the infection from the acute to the chronic type.

The symptoms are similar to those found in the acute form. The chicks that are affected are older and the losses are not as large.

Lesions are present in a large percentage of instances. The lungs, gizzard, liver and heart are the organs in which they are found. The microscopic appearance of these lesions is reserved for further study. The fact that they have been found in a large percentage of infections and bacteriological examination has shown *Bacterium pullorum* to be present, allows attention to be called to their diagnostic importance.

The liver, the surface of which is exposed to view upon opening the peritoneal cavity, may show these foci as pin-point, white areas. Slicing the organ reveals their presence throughout the organ. Removing the liver, gizzard, spleen and intestines bares the ventral surface of the lungs. White foci, rather firm in consistency, may been seen. (Fig. 5)

The lungs should be carefully dissected from the thorax, using a blunt instrument, as lesions visible on the dorsal surface may be found. (Fig. 4) The ventral surface of the lungs may not show these foci and unless the lungs are carefully removed and examined. their presence elsewhere may be overlooked.

A careful examination of the contour of the gizzard may reveal raised areas, 1 to 2 mm. in diameter. When cut into they are found to vary from moderately firm foci to apparently necrotic areas. When these raised areas are not detected, the gizzard muscle should be cut into by making several incisions. Foci deep in the muscles will then usually be found. (Fig. 4)

The ventricles of the heart may appear irregular. A more careful examination shows a thickened condition of the heart muscle, the thickened portions being much lighter in color than the normal part.

Unabsorbed yolk sacs are not generally found. When they are found, the yolk capsule is rather tough. The yolk material has lost its "true" yolk consistency. In older chicks the presence of an unabsorbed yolk sac is more indicative of pullorum infection than it is in young ones. Its presence, however, cannot be taken as a definite indication of pullorum infection.

Treatment, chicks. When an outbreak of bacillary white diarrhea occurs among chicks, the owner wishes to save as many as possible. This is naturally so, but the fact that the survivors may become "carriers" of the disease must be kept in mind. The survivors may be reared but should not be used later for breeding purposes. The general recommendation of disposing of the survivors as soon as a diagnosis is made, or rearing them for market, will be found to be the safest in the end.

Acidophilus milk, or mild disinfectants placed in the drinking water, are supposed to have some effect in checking the disease. An

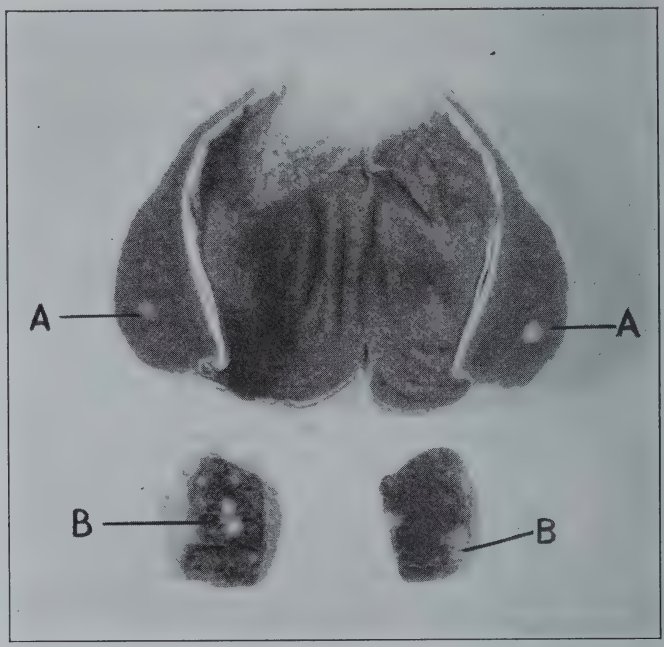


Fig. 4. Necrotic foci: A, in the muscle of the gizzard; B, in the lungs.

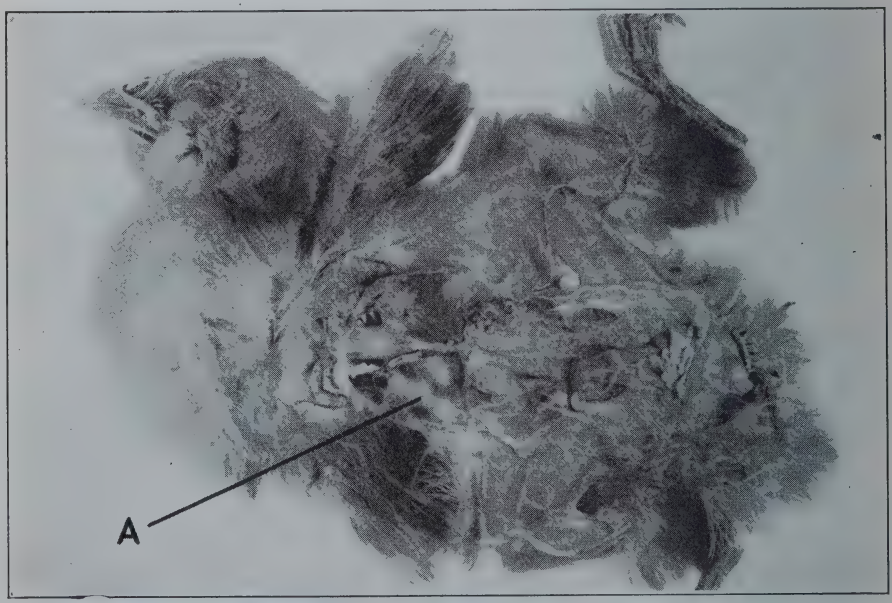


Fig. 5. Necrotic foci in the lungs.

attempt is made in using the above treatments to prevent multiplication of the ingested bacteria in the intestinal tract and their subsequent invasion of the tissues. Chicks already infected when hatched would not be benefited, but uninfected chicks in the same brooder might possibly be protected.

Treatment, adults. There is no satisfactory treatment for adults. The difficulty in removing the infection from the ovary or other infected tissues is readily apprehended.

Eradication. The three chief sources of the infection for chicks has been shown to be the adult fowls, infected eggs, and contaminated brooders.

The contaminated brooders are easily disposed of by using only properly disinfected brooders for the chicks. The fact that the organism is easily destroyed by the common disinfectants does not make this at all difficult.

The source of the infection through the egg can be eliminated by using eggs only from hens that are not infected. This leaves the chief source of infection with the adult breeder.

Jones (5) has shown that there are present in the serum of infected adults, agglutinins which may readily be detected by the macroscopic test. Another test, the intradermic wattle test, although not used very extensively at present, does appear to have some merits. The two tests are discussed briefly.

Intradermic wattle test. The antigen or test fluid consists of the bacteria-free filtrate of the bouillon in which *Bact. pullorum* has been allowed to grow for a period of 4 to 12 weeks. The filtrate is concentrated, and 0.5 per cent phenol is added for preserving purposes. The injection is made into either wattle, using an intradermic needle. Approximately 0.2 cc. of the fluid is injected. Readings are taken at the end of 24 and 30 hours. A reaction consists of an edematous swelling of the wattle which may be compared with the uninjected wattle. Because of the fact that an antigen has not as yet been successfully made which will give satisfactory readings, this method has not been extensively used.

Macroscopic agglutination test. In testing a flock for the determination of "carriers," the work may be grouped under the following headings:

1. Preparation of the flock.
2. Collection of the blood.
3. Care of the blood.
4. Preparation of the antigen.
5. Setting the tests.
6. Reading the tests.
7. Follow up measures.

1. *Preparation of the flock.* Hitchner(7) has shown that the blood from flocks in heavy production, or from flocks which have been fed shortly before bleeding, will contain a sufficient amount of so-called fat to interfere with an accurate reading of the tests. In order to obtain blood serum which will not contain this so-called

fatty material, it is necessary to starve the birds previous to collecting the blood samples. The 24-hour starvation period is rather a strenuous procedure on a flock in production. The length of time does not appear to be necessary. The day before the blood samples are to be taken the mash is taken away from the flock at noon and the scratch grain is omitted at night. The mash and grain are placed before them again soon after bleeding. This method does not decrease egg production as much as the 24-hour period of starvation. Some decrease in egg production due to this starvation period and the handling of the hens, must be expected.

The preferable time to collect the blood samples is, of course, when egg production is low and the flock is being assembled for winter quarters. Non-laying hens or ones in low production furnish satisfactory serum without the necessity of a starvation period.

All of the chickens to be tested must, of course, be legbanded. The numbers and type of the band should be such that the work will be aided, and not hindered, by them.

2. *Collection of the blood.* The blood is collected as illustrated (Figs. 6 and 7) from the large vein as it passes over the first wing joint (humeroradial) from the body on the under side. The fowl is held by the legs and wings on its side on a barrel top or table. The upper wing is spread out. The small feathers over the joint are plucked out. The vein will readily be seen. Placing the finger on the vein, between the joint and heart, as it passes along the humerus, will cause it to be distended. In some fowls this may be necessary, but in most it is not. An incision is made with a scalpel (a half inch blade) either across the vein or longitudinally with it. The blood will flow out through the wound and may be collected in the bottle. Approximately 2 cc. of the blood is sufficient. The bottles should be tilted and the blood allowed to coagulate in a slanting position, as illustrated. (Fig. 8) Fowl blood coagulates very quickly, which necessitates placing each bottle in a slanting position as soon as it is filled.

3. *Care of the blood.* The blood should not be allowed to freeze or become overheated. It should be kept cool enough to keep down bacterial multiplication. The above factors, if allowed to occur, will hemolyze the blood cells and the serum will become reddish, and will not be as satisfactory to use as the clear straw-colored serum which is desired. The slanted bottles, if allowed to stand upright in a cool place overnight, will contain enough serum at the lower part of the slant for the test the next day. (Fig. 8-A) If a sufficient amount of serum has not collected, the blood clot may be broken up, using a wire rod. The rod should be rinsed and dried each time it is used to prevent the transferring of blood from one sample to another. The bottles are then allowed to stand in a cool place again overnight. Clear, straw-colored serum should be found in the morning. If the serum is still lacking, centrifuging of the blood is then necessary.

Fig. 6. Making the incision.

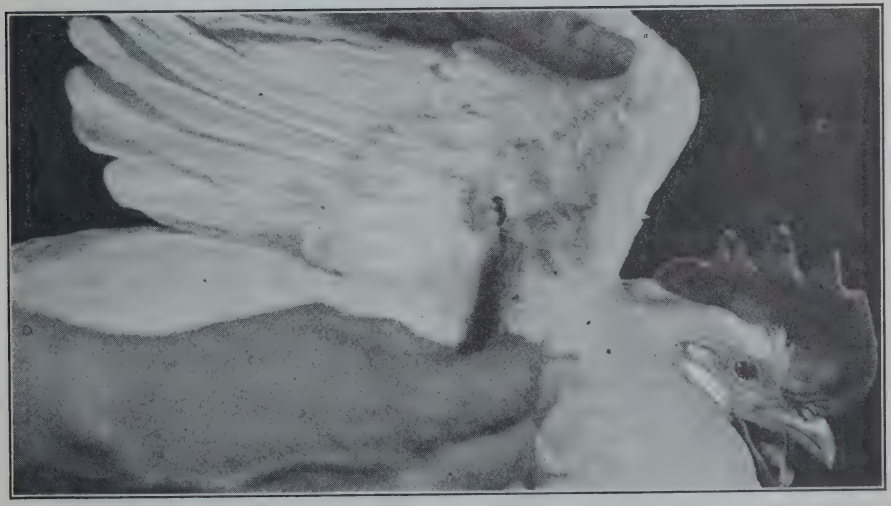


Fig. 7. Collecting the blood.

4. *Preparation of the antigen.* The antigen consists of a suspension of one or more strains of *Bact. pullorum* which are agglutinable. This is prepared by seeding agar slants with strains of the organisms to be used. After 24 hours' incubation at 37.50°C, the growth is washed off using sterile physiological salt solution. The antigen is diluted until the desired turbidity is obtained. A quantity of the antigen can be prepared and kept for a period of weeks when stored in a cool place in an uncontaminated condition.

5. *Setting the tests.* One or two dilutions of the serum may be used for the test. There seems to be a difference of opinion at this writing as to the dilution of the serum and the number of dilutions which shall be used in routine testing. This laboratory

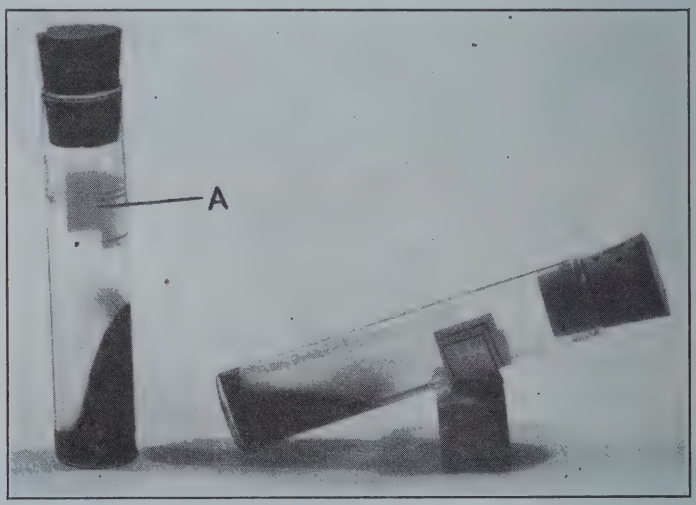


Fig. 8. Slanting of blood sample.

has evidence that adult chickens whose serum has a titre of 1:40 may show ovarian infection. Whether or not this dilution is the critical one, remains as yet to be determined. The question of a proagglutinating zone is one which cannot be entirely ignored. The number of individuals showing this peculiarity of the serum are perhaps few. In order to detect these individuals a higher dilution of the serum is necessary.

At the present writing two dilutions of the serum, 1:40 and 1:60, are being used. Test tubes 100 mm. long and 10 mm. wide have been found to be satisfactory. One-twentieth of a mil of the clear serum is pipetted into each of two tubes. Two mils of the antigen is added to one of the tubes diluting the serum 1:40. Three mils of the antigen is added to the other tube making a second dilution of the serum 1:60. The serum and antigen are mixed by shaking the tubes.

The serum identification number may be copied on the test tubes using a wax pencil or a label with the number may be attached to them. It is rather time consuming to identify the tests in this manner and we have found the use of wooden blocks very satisfactory. A block 10¼ inches long, 4¾ inches wide and one inch thick is perforated with sixty holes large enough to accommodate the test tubes, the holes being arranged in rows about one-quarter of an inch apart. A false bottom is fastened to the block. As each block holds sixty tubes or thirty tests there is a considerable saving of space in the incubator.

The pipette is rinsed out with clean water after the handling of each samples of serum. A large flask of water conveniently near the operator makes this an easy operation.

Two control tubes containing antigen alone are placed with the tests. Spontaneous agglutination of the antigen should be detected by them. They will also serve as a comparison with the other tests.

The tests completed, they are then placed in an incubator or in some other suitable place where a temperaature of 37.5°C is maintained. Overnight incubation is sufficient. Placing the tests for a day in an icebox after incubation gives more definite readings. The tests can be read without this extra waiting period, but are not as satisfactory.

6. *Reading the tests.* The reaction of the serum on the antigen may be grouped under three heads as follows:

Positive reaction. The column of turbid antigen will have become entirely clear. The bottom of the tube will be covered with a "blanket" of the agglutinated bacteria. If the tube is shaken, the "blanket" will go into suspension but it will appear more "granular" than the original.

Negative reaction. There is no change in the turbidity of the antigen. It appears the same as when first added to the tubes. The control tubes and negative reactions should appear alike. A careful examination of the bottom of the tube will show a "button." This "button" is more compact and much smaller than the "blanket" mentioned as occurring in positive tests. The settling of some of the bacteria is supposed to account for its presence.

Suspicious reaction. The tubes may contain a "blanket" on the bottom, but the column of antigen has not become clear. This may be due to the use of too heavy an antigen; that is, too many bacteria in suspension. This is remedied by diluting the antigen and resetting the tests.

If the antigen is sufficiently dilute and some agglutination is evidenced by a blanket formation, but the column of antigen has not become clear, the serum should be tested at a lower dilution. Sera which give such reactions at a dilution of 1:40, may be found to have a complete agglutinating titre of 1:20.

The tubes may be turbid but contain flaky material. This may be present at the top of the column of antigen and suggest something of a fatty nature. Other material, not fat-like, may be

present in the lower part of the tube. It does not settle completely on the bottom but remains in suspension.

The blood sera of laying fowls are apt to give confusing tests, the material preventing a satisfactory reading of the test being fat-like as previously described. Starving the birds, as recommended, seems to eliminate this factor.

The second type of material which confuses the accurate reading of the tests, might be due to another cause. Rebrassier(8) states that the use of fresh, unheated, unpreserved antigen has eliminated trouble of this nature. This pseudo-agglutination, as it might be termed, was present in a number of their tests when phenol was added to the antigen as a preservative. Omitting the phenol removed this difficulty.

7. *Follow-up measures.* In flocks where the agglutination test is being used to eradicate the disease, the poultryman must institute certain rules in order that the work be a success. Reacting fowls should be removed from the premises as soon as possible. If it is desired to retain them a few months for egg production, they should be maintained as a separate flock. This is often rather difficult to do and encouragement should *not* be given to this plan, at least not until we are better informed regarding certain phases of the disease in adults. The houses and feeding utensils should be disinfected after the reactors have been removed.

The agglutination test should be repeated each year, or each half year, until no reactors are found. With the establishment of a flock free from the infection, care must be taken so that it will remain so. The chief ways, insofar as we know, by which the infection can be reintroduced, are adult stock, baby chicks and eggs. In purchasing baby chicks and eggs, the owner should buy only from flocks free from the disease. If the owner is not certain about the flock from which the baby chicks or eggs came, they should be kept isolated until they can be tested. All adult stock should be tested and found not to react to the agglutination test before being added to the flock.

Continuous yearly testing of the flock after all infected individuals have been removed, may not be practical from the owner's standpoint. Although it may seem to be the most ideal way to control this disease at the present time, the financial obligation might be too great. It is important though that every precaution be taken against reintroducing the infection.

BIBLIOGRAPHY

1. JONES, F. S., Fatal Septicemia or Bacillary White Diarrhea in Young Chickens. Annual Report of the New York State Veterinary College, 1909–10.
2. RETTGER, L. F., KIRKPATRICK, WM. F., AND JONES, R. E., Bacillary White Diarrhea of Young Chicks (fiftieth report), Bulletin No. 85, December, 1915, Storrs Agric. Exp. Sta.
3. DOYLE, T. M., Bacillary White Diarrhea of Chicks. Jour. of Comparative Pathology and Therapeutics. Vol. 38, No. 4, December 31, 1925.
4. BEAUDETTE, BUSHNELL, AND PAYNE, The Relation of *Bact. pullorum* to Hatchability of Eggs, J. Inf. Dis., xxxiii, 1923.
5. JONES, F. S., An Outbreak of an Acute Disease in Adult Fowls due to *Bact. pullorum;* The Value of the Macroscopic Agglutination Test in Detecting Fowls that Are Harboring *Bact. pullorum,* Annual Report of the New York State Veterinary College, 1911–12.
6. HINSHAW, W. R., UPP, C. W., AND MOORE, J. M., Studies in Transmission of Bacillary White Diarrhea in Incubators. J. A. V. M. A., New Series, Vol. 21, No. 5, February, 1926.
7. HITCHNER, E. R., The Macroscopic Agglutination Test as Influenced by the Fatty Content of the Blood Serum of Fowls. J. A. V. M. A., New Series, 1923, xvi, 759.
8. REBRASSIER, R. E. Studies of Salmonella pullora. J. A. V. M. A., New Series, Vol. 21, No. 5, February, 1926.

THE DIFFERENTIATION OF BACT. PULLORUM (RETTGER) AND BACT. SANGUINARIUM (MOORE)

J. M. HENDRICKSON

Department of Pathology and Bacteriology

In 1889 E. Klein(8) isolated an organism from chickens that died during an epidemic in Kent, England, which he called *Bact. gallinarum.* The description he gives of this organism, in regard to morphology, staining and cultural characteristics, and inoculation experiments, is very similar to that given by Moore(9) of an organism that he isolated from an epidemic among fowls in this country in 1895, which he called *Bact. sanguinarium,* and which has since been considered to be the causative agent of fowl typhoid. Hadley(3) in 1918 showed that these two organisms were identical.

In 1900 Rettger(10) isolated from chicks an organism which he later proved was the causative agent of bacillary white diarrhea. This organism was morphologically and culturally very similar to *Bact. sanguinarium* except that it produced gas in certain carbohydrate media while the latter produced only acid. Also there appeared to be certain differences in the carbohydrates fermented by these two organisms.

In 1913 Jones (7) described an acute disease in adult fowls due to *Bact. pullorum.* This organism differed from that described by Rettger in that it did not produce gas in any carbohydrate, but in all other respects it appeared to be identical with it. However, there are no data given in Jones' article that would differentiate his organism from *Bact. sanguinarium.* Hadley(4) also reports infections in adult fowls due to this non-gas producing type. Later work has shown these two types of organisms to be identical except in the matter of gas production. The gas producing type has become known as *Bact. pullorum A* and the non-gas producing as *Bact. pullorum B.*

The differentiation of the non-gas forming type of *Bact. pullorum* from *Bact. sanguinarium* is a problem that is very frequently met with in the routine laboratory diagnosis of poultry diseases. A great deal of work has been done in the differentiation of these organisms by carbohydrate fermentation with the result that *Bact. sanguinarium* is usually considered to be maltose, dextrin, dulcit positive while *Bact. pullorum B* fails to ferment these sugars(2, 3). Serologically it has been found that there is a very close relationship by the cross agglutination of these organisms and that *Bact. pullorum* immune serum will agglutinate the sanguinarium organism about as well as it will the pullorum organism and vice versa (3, 11). Smith and Ten Broeck (12) state that toxin production is identical and differences in immunological reaction have not been found.

A number of strains have recently been isolated from chicks in this laboratory that have failed to produce gas in the sugars which

are ordinarily fermented by the pullorum organisms. This is unusual, as it generally has been found that the gas producing type is the cause of infections in young chicks and the non-gas producing strains are the etiological factors in adult infections. Also a number of these non-gas producing strains have produced acid in maltose extract bouillon on over-night incubation.

Thinking that the media might be at fault, several new lots were prepared and used, with the same results. Other types of gas producing organisms have produced gas in all lots regularly. Extract bouillon with the addition of approximately one per cent sugar has been used in the routine work but in a few cases infusion bouillon has been tried with the same results.

It was also noted that stock strains of *Bact. pullorum,* both of the A and B type, would produce acid in maltose extract bouillon if the cultures were allowed to stand about the laboratory for some time. This, together with the fact that some of the freshly isolated non-gas producing strains produced acid in maltose extract bouillon on over-night incubation, indicated that these strains were either *Bact. sanguinarium,* or that maltose could not be used as a means of differentiation between this organism and *Bact. pullorum B.* However, none of the pullorum strains fermented dextrin while all of the stock sanguinarium strains readily did so.

The fact that maltose is a rather unstable sugar and easily breaks down into dextrose was considered, but several lots of maltose were used with the same results. To prevent this breaking down, the maltose, and in fact all sugars, were autoclaved in ten per cent solution in distilled water and then pipetted into the sterile bouillon in sufficient quantity to make approximately a one per cent sugar solution.

These observations on the irregularities of these organisms showed that the present methods of differentiating them on carbohydrate fermentation were inadequate, and this work was undertaken in the attempt to find some definite means of differentiation. The work naturally divided itself into:

1. Fermentation studies.
2. Serological studies.

Fermentation Studies

It has been shown by Doyle and Spray(13) that the pullorum organisms would ferment maltose quite readily in serum water. Broadhurst(1) brought out in her work with streptococci that the kind of media used had a marked effect on the fermentation power of these organisms and that the optimum nutrient medium for the organisms concerned should always be used in work of this character. She found that streptococci produced two or three times as much acid when grown in meat infusion as in meat extract media. Jones(6) showed this again, and also that the addition of a little blood serum to infusion or extract bouillon markedly increased acid production. This suggested that perhaps in a more favorable medium, such as serum water, carbohydrates that remained un-

changed in the ordinary infusion or extract bouillon might be attacked. In this work serum water, infusion bouillon from meat, and extract bouillon were used.

PREPARATION OF MEDIA

The serum water was prepared by adding ox serum to distilled water in the proportion of one part serum to three of water. One per cent Andrade's indicator was added to this and then it was tubed and autoclaved.

The infusion bouillon was prepared from meat (ground beef) in the ordinary way and then rendered sugar free by spontaneous anaerobic fermentation. It was then titrated and the reaction adjusted to 0.2 per cent acid to phenolphthalein. One per cent Andrade's indicator was added and the bouillon autoclaved. Samples of the bouillon were tested for the presence of sugar in fermentation tubes with pure cultures in *Bact. coli* and found sugar free.

The extract bouillon was prepared with 0.2 per cent meat extract, 1 per cent peptone, and 0.5 per cent NaCl. The reaction was adjusted to 0.2 per cent acid to phenolphthalein, 1 per cent Andrade's indicator was added and the bouillon autoclaved.

Fourteen carbohydrates were used. They were all put up separately in 10 per cent solution in distilled water, autoclaved and then pipetted into the various sterile tubes of media in amount to make approximately a 1 per cent solution.

ORGANISMS USED

Most of the organisms were stock strains that had been carried in the laboratory for some time. Some recently isolated cultures of pullorum A were used because only one of the stock strains of that type would produce gas at this time in carbohydrate media. These strains had apparently lost their power of gas production under continued artificial cultivation. Several lots of each type of media and several lots of sugars were tried, always with the same results. Some of these cultures were incubated at 37°C as long as one month.

HISTORY OF STRAINS USED

Bact. sanguinarium (Moore) was obtained from Dr. F. R. Beaudette of the New Jersey Agricultural Experiment Station and is supposedly a sub-culture of the original strain isolated by Moore (9) in 1895.

Bact. gallinarum (Klein) was also obtained from Dr. F. R. Beaudette and is supposedly a sub-culture of the original strain isolated by Klein (8) in 1889.

Bact. sanguinarium (Smith) was a stock strain originally obtained from Dr. Theobold Smith.

Bact. sanguinarium is an old stock culture on which it was impossible to obtain any history.

Bact. sanguinarium (Taylor) was obtained from Dr. W. G. Taylor previous to 1917. This strain apparently has lost all power of fermenting carbohydrates. Morphologically and in staining char-

acteristics it appeared to be typical but it consistently failed to ferment carbohydrates. All three types of media were tried and some of the tubes were incubated for as long as two months.

Pullorum B Strains

Number 1364 was isolated June 5, 1924, from a dead chick received at the laboratory for diagnosis.

Number 1128 was isolated December 14, 1923, from the internal organs of a hen. This bird was sent into the laboratory with the history that thirty birds had died in a short period of time. This organism produced acid in maltose and dextrose on isolation. Dextrin and dulcit were not tried.

Number 1150 was isolated on January 21, 1924, from the ova of a hen.

Number 1343 was isolated May 28, 1924, from a baby chick.

Number 1153 was isolated January 22, 1925, from the diseased ova of a hen.

Pullorum A Strains

Number 1791 was isolated April 2, 1925, from the liver of a baby chick.

Number 1454 was isolated July 28, 1924, from the liver of a 2½ month old chick.

Number 1805 was isolated April 8, 1925, from the liver of a baby chick. This organism produced acid in maltose on over-night incubation when first isolated.

Number 1822 was isolated April 14, 1925, from a baby chick.

Number 1803 was isolated April 8, 1925, from a baby chick. It produced acid in maltose on over-night incubation when first isolated.

Fermentation of Carbohydrates

In the fermentation tests fourteen carbohydrates were used. The various strains were transferred daily for from three to five days, or more, in serum water or infusion bouillon to get them to growing vigorously. The carbohydrate media were then inoculated from these cultures with Pasteur pipettes. Some of the tubes, no doubt, received a larger amount of inoculum than others and this may account for some of the delayed reactions during the first period of incubation. A seal was put into all tubes by pipetting melted vaseline into them. This formed a tight seal over the top of the medium and when gas was formed this seal was pushed up in the tube. Readings were made every day for two weeks, and then twice weekly up to forty-four days. Only a limited number of these readings are reported in the tables as the slight changes that occurred from day to day after the first few days did not warrant the making of the large table that would be necessary to include all these data.

Summary of Fermentation Tests

Dextrose

This sugar was fermented about equally well by all strains.

TABLE No. 1

PULLORUM A — CULTURE No. 1454

	1st Day			2d Day			4th Day			6th Day			16th Day			26th Day			44th Day		
	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract
Dextrose	+ +	+ −	+ B	+ +	+ +	+ +	+ +	+ +	+ +	+ +	+ +	+ +	+ +	+ +	+ +	+ +	+ +	+ +	+ +	+ +	+ +
Lactose	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −
Sucrose	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −
Maltose	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	+ −	+ −
Mannite	+ −	+ −	+ −	+ B	+ B	+ −	+ B	+ B	+ −	+ B	+ B	+ B	+ B	+ B	+ B	+ B	+ B	+ B	+ B	+ B	+ B
Dextrin	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −
Dulcit	− −	− −	− −	− −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −
Isodulcit	+ B	+ B	+ −	+ B	+ B	+ B	+ B	+ B	+ B	− B	+ B	+ B	− B	+ B	+ B	− B	+ B	+ B	− B	+ B	+ B
Xylose	− −	− −	− −	+ −	+ −	− −	+ −	+ −	− −	+ B	+ B	+ B	+ B	+ B	+ B	+ B	+ B	+ B	+ B	+ B	+ B
Salicin	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −
Raffinose	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −
Inulin	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −
Galactose	+ B	+ B	+ −	+ +	+ +	+ B	+ +	+ +	+ B	+ +	+ +	+ B	+ +	+ +	+ B	+ +	+ +	+ B	+ +	+ +	+ B
Arabinose	+ B	+ B	− −	+ +	+ +	+ −	+ +	+ +	+ −	+ +	+ +	+ −	+ +	+ +	+ −	+ +	+ +	− −	+ +	+ +	+ −

+ + Acid and gas. + − Acid only. − − Negative. − + Gas only. + B Acid and bubble of gas.

TABLE No. 2

PULLORUM A — CULTURE No. 1805

	1st Day			2d Day			4th Day			6th Day			16th Day			26th Day			44th Day		
	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract
Dextrose	+ +	+ B	+ —	+ +	+ +	+ —	+ +	+ +	+ —	+ +	+ +	+ —	+ +	+ +	+ —	+ +	+ +	+ B	+ +	+ +	+ B
Lactose	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —
Sucrose	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —
Maltose	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	+ —	+ —
Mannite	+ —	+ —	+ —	+ B	+ —	+ —	+ B	+ —	+ —	+ B	+ —	+ —	+ B	+ —	+ —	+ B	+ B	+ B	+ +	+ B	+ B
Dextrin	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —
Dulcit	— —	— —	— —	— —	— —	— —	— —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —
Isodulcit	+ —	+ —	— —	+ B	+ B	+ —	+ B	+ B	+ —	— +	+ +	+ —	— +	+ +	+ —	— +	+ +	+ —	— +	+ +	+ —
Xylose	+ —	— —	— —	+ —	— —	— —	+ —	+ —	— —	+ —	+ B	— —	+ B	+ B	+ —	+ B	+ B	+ —	+ B	+ B	+ —
Salicin	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —
Raffinose	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	+ —	+ —
Inulin	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	+ —	+ —
Galactose	+ —	+ —	+ —	+ B	+ B	+ —	+ B	+ B	+ —	+ B	+ B	+ —	+ B	+ B	+ —	+ B	+ B	+ —	+ B	+ B	+ —
Arabinose	+ —	+ —	+ —	+ B	+ B	+ —	+ B	+ B	+ —	+ B	+ B	+ —	+ B	+ B	+ —	+ B	+ B	+ —	+ B	+ B	+ —

+ + Acid and gas. + — Acid only. — — Negative. — + Gas only. + B Acid and bubble of gas.

TABLE No. 3

PULLORUM A — CULTURE No. 1803

	1st Day			2d Day			4th Day			6th Day			16th Day			26th Day			44th Day		
	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract
Dextrose	+ B	+ B	+ −	+ B	+ B	+ B	+ B	+ B	+ B	+ B	+ B	+ B	+ B	+ B	+ B	+ B	+ B	+ B	+ B	+ B	+ B
Lactose	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −
Sucrose	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −
Maltose	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	+ −	+ −	+ −	+ −
Mannite	+ −	+ −	+ −	+ B	+ B	+ −	+ B	+ B	+ −	+ B	+ B	+ −	+ B	+ B	+ −	+ B	+ B	+ −	+ B	+ B	+ −
Dextrin	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −
Dulcit	− −	− −	− −	− −	− −	− −	− −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −
Isodulcit	+ +	+ B	+ −	+ +	+ B	+ −	+ +	+ B	+ −	+ +	+ B	+ −	+ +	+ B	+ −	+ +	+ B	+ −	+ +	+ +	+ B
Xylose	+ −	− −	− −	+ −	− −	− −	+ −	+ −	− −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −
Salicin	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −
Raffinose	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	+ −	− −
Inulin	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	+ −
Galactose	+ B	+ B	+ −	+ +	+ B	+ B	+ +	+ +	+ B	+ +	+ +	+ B	+ +	+ +	+ B	+ +	+ +	+ B	+ +	+ +	+ B
Arabinose	+ +	+ B	+ −	+ +	+ B	+ −	+ +	+ +	+ −	+ +	+ +	+ −	+ +	+ +	+ −	+ +	+ +	+ −	+ +	+ +	+ −

+ + Acid and gas. + − Acid only. − − Negative. − + Gas only. + B Acid and bubble of gas.

TABLE No. 4

PULLORUM A — CULTURE No. 1791

	1st Day			2d Day			4th Day			6th Day			16th Day			26th Day			44th Day		
	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract
Dextrose	+ +	+ −	+ −	+ +	+ +	+ −	+ +	+ +	+ −	+ +	+ +	+ −	+ +	+ +	+ −	+ +	+ +	+ −	+ +	+ +	+ −
Lactose	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −
Sucrose	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −
Maltose	− −	− −	− −	− −	− −	− −	− −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	+ −	+ −	+ −	+ −	+ −
Mannite	+ +	+ −	+ −	+ +	+ −	+ −	+ +	+ −	+ −	+ +	+ +	+ −	+ +	+ +	+ −	+ +	+ +	+ B	+ +	+ +	+ B
Dextrin	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −
Dulcit	− −	− −	− −	− −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −
Isodulcit	+ +	+ −	− −	+ +	+ +	+ −	+ +	+ +	+ −	− +	+ +	+ −	− +	+ +	+ −	− +	+ +	+ −	− +	+ +	+ B
Xylose	+ −	− −	− −	+ −	− −	− −	+ +	+ +	+ −	+ +	+ +	+ +	+ +	+ +	+ +	+ +	+ +	+ +	+ +	+ +	+ +
Salicin	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −
Raffinose	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	+ −	− −
Inulin	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	+ −	+ −
Galactose	+ +	+ +	+ −	+ +	+ +	+ B	+ +	+ +	+ B	+ +	+ +	+ B	+ +	+ +	+ B	+ +	+ +	+ B	+ +	+ +	+ B
Arabinose	+ +	+ +	+ −	+ +	+ +	+ −	+ +	+ +	+ −	+ +	+ +	+ −	+ +	+ +	+ −	+ +	+ +	+ −	+ +	+ +	+ −

+ + Acid and gas. + − Acid only. − − Negative. − + Gas only. + B Acid and bubble of gas.

TABLE No. 5

PULLORUM A.

CULTURE No. 1822

	1st Day			2d Day			4th Day			6th Day			16th Day			26th Day			44th Day		
	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract
Dextrose	+ B	+ B	+ —	+ B	+ B	+ —	+ B	+ B	+ B	+ B	+ B	+ B	+ B	+ B	+ B	+ B	+ B	+ B	+ B	+ B	+ B
Lactose	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —
Sucrose	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	+ —	— —	— —	+ —	+ —	— —
Maltose	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —
Mannite	+ B	+ —	+ —	+ B	+ B	+ —	+ B	+ B	+ —	+ B	+ B	+ —	+ B	+ B	+ —	+ B	+ B	+ —	+ B	+ B	+ B
Dextrin	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —
Dulcit	— —	— —	— —	— —	— —	— —	— —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —
Isodulcit	+ B	+ B	— —	+ +	+ +	— —	+ +	+ +	+ —	+ +	+ +	+ —	— +	+ +	+ —	— +	+ +	+ —	— —	— —	+ +
Xylose	+ —	— —	— —	+ —	— —	— —	+ —	+ —	— —	+ +	+ +	+ —	+ +	+ +	+ +	+ +	+ +	+ —	+ +	+ +	+ +
Salicin	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —
Raffinose	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —
Inulin	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —
Galactose	+ +	+ +	+ —	+ +	+ +	+ B	+ +	+ +	+ B	+ +	+ +	+ B	+ +	+ +	+ B	+ +	+ +	+ B	+ +	+ +	+ B
Arabinose	+ +	+ +	+ —	+ +	+ +	+ —	+ +	+ +	+ —	+ +	+ +	+ —	+ +	+ +	+ —	+ +	+ +	+ —	+ +	+ +	+ —

+ + Acid and gas. + — Acid only. — — Negative. — + Gas only. + B Acid and bubble of gas.

TABLE No. 6

PULLORUM B — CULTURE No. 1153

	1st Day			2d Day			4th Day			6th Day			16th Day			26th Day			44th Day		
	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract
Dextrose	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —
Lactose	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —
Sucrose	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	+ —	+ —	— —
Maltose	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	+ —	+ —	+ —	— —	— —	+ —	+ —	— —
Mannite	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	— —	+ —	+ —	+ —	+ —	+ —	— —	+ —
Dextrin	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —
Dulcit	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —
Isodulcit	+ —	+ —	— —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —
Xylose	+ —	— —	— —	+ —	— —	— —	+ —	+ —	— —	+ —	+ —	— —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	— —
Salicin	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	+ —	— —	— —	— —	— —	— —
Raffinose	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —
Inulin	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —	+ —	— —	— —
Galactose	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —
Arabinose	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —

++ Acid and gas. +— Acid only. —— Negative. —+ Gas only.

TABLE No. 7

PULLORUM B — CULTURE No 1128

	1st Day			2d Day			4th Day			6th Day			16th Day			26th Day			44th Day		
	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract
Dextrose	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −
Lactose	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −
Sucrose	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −
Maltose	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	+ −	+ −	+ −	+ −
Mannite	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −
Dextrin	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −
Dulcit	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −
Isodulcit	+ −	+ −	− −	+ −	+ −	+ −	+ −	+ −	+ −	− −	+ −	+ −	− −	+ −	+ −	− −	− +	+ −	− −	+ −	+ −
Xylose	− −	− −	− −	− −	− −	− −	+ −	+ −	+ −	+ −	+ −	− −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −
Salicin	− −	− −	− −	− −	− −	− −	− −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −
Raffinose	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −
Inulin	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	+ −	− −
Galactose	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −
Arabinose	+ −	+ −	− −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −

++ Acid and gas. +− Acid only. −− Negative. −+ Gas only.

TABLE No. 8

PULLORUM B

CULTURE No. 1364

	1st Day			2d Day			4th Day			6th Day			16th Day			26th Day			44th Day		
	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract
Dextrose	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −
Lactose	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −
Sucrose	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −
Maltose	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −
Mannite	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	− −	− −	+ −	+ −	+ −	+ −	− −	+ −
Dextrin	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −
Dulcit	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	− −	+ −	+ −	− −	− −	− −	− −	+ −	+ −	+ −	+ −	+ −
Isodulcit	+ −	+ −	− −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −
Xylose	+ −	− −	− −	+ −	+ −	− −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	− −	− −	− −	− −	− −	− −
Salicin	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −
Raffinose	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	+ −	+ −
Inulin	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −
Galactose	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −
Arabinose	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −

+ + Acid and gas. + − Acid only. − − Negative. − + Gas only.

TABLE No. 9

PULLORUM B — CULTURE No. 1150

	1st Day			2d Day			4th Day			6th Day			16th Day			26th Day			44th Day		
	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract
Dextrose	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −
Lactose	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −
Sucrose	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −
Maltose	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −
Mannite	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −
Dextrin	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −
Dulcit	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −
Isodulcit	+ −	+ −	− −	+ −	+ −	+ −	− −	+ −	+ −	− −	+ −	+ −	− −	+ −	+ −	− −	− −	+ −	− −	− −	+ −
Xylose	+ −	− −	− −	+ −	+ −	− −	+ −	+ −	− −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −
Salicin	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −
Raffinose	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −
Inulin	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −
Galactose	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −
Arabinose	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −

++ Acid and gas. +− Acid only. −− Negative. −+ Gas only.

TABLE No. 10

PULLORUM B — CULTURE No. 1343

	1st Day			2d Day			4th Day			6th Day			16th Day			26th Day			44th Day		
	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract
Dextrose	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −
Lactose	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −
Sucrose	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −
Maltose	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −
Mannite	+ −	+ −	− −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −
Dextrin	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −
Dulcit	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −
Isodulcit	+ −	+ −	− −	+ −	+ −	+ −	− −	+ −	+ −	− −	+ −	+ −	− −	+ −	+ −	− −	+ −	+ −	− −	− +	+ −
Xylose	− −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −
Salicin	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −
Raffinose	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −
Inulin	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −
Galactose	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −
Arabinose	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −

++ Acid and gas. - - +− Acid only. −− Negative. −+ Gas only.

TABLE No. 11

SANGUINARIUM MOORE

	1st Day			2d Day			4th Day			6th Day			16th Day			26th Day			44th Day		
	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract
Dextrose	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −
Lactose	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −
Sucrose	− −	− −	− −	− −	− −	− −	− −	− −	− −	+ −	+ −	+ −	+ −	+ −	+ −	− −	+ −	+ −	− −	+ −	+ −
Maltose	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −
Mannite	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	− −	+ −	+ −	− −	+ −	+ −
Dextrin	− −	− −	− −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −
Dulcit	+ −	− −	− −	+ −	+ −	+ −	+ −	+ −	+ −	− −	+ −	+ −	+ −	+ −	+ −	− −	+ −	+ −	− −	+ −	+ −
Isodulcit	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −
Xylose	+ −	+ −	− −	+ −	+ −	+ −	+ −	+ −	− −	− −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −
Salicin	− −	− −	− −	− −	− −	− −	− −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	+ −	+ −
Raffinose	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	+ −	+ −
Inulin	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −
Galactose	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −
Arabinose	+ −	− −	− −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	− −	− −	+ −	− −	− −	+ −	+ −	+ −

++ Acid and gas. +− Acid only. −− Negative. −+ Gas only.

TABLE No. 12

SANGUINARIUM SMITH

	1st Day			2d Day			4th Day			6th Day			16th Day			26th Day			44th Day		
	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract
Dextrose	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+	+	+ −
Lactose	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	−	−	− −
Sucrose	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	−	−	− −
Maltose	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −
Mannite	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −
Dextrin	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −
Dulcit	+ −	− −	− −	+ −	+ −	− −	+ −	+ −	− −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −
Isodulcit	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	+ −	− −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −
Xylose	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −
Salicin	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	−	−	− −
Raffinose	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	+ −	− −
Inulin	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	+ −	− −
Galactose	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −
Arabinose	+ −	+ −	− −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −

+ + Acid and gas. + − Acid only. − − Negative. − + Gas only.

TABLE No. 13

B. SANGUINARIUM

	1st Day			2d Day			4th Day			6th Day			16th Day			26th Day			44th Day		
	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract
Dextrose	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −
Lactose	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −
Sucrose	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	+ −	+ −	+ −	− −	+ −	+ −	− −	+ −	+ −
Maltose	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −
Mannite	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	− −	+ −	− −	+ −	+ −	− −	+ −	+ −	− −	+ −	+ −
Dextrin	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −
Dulcit	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −
Isodulcit	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −
Xylose	+ −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	+ −	+ −	+ −
Salicin	− −	− −	− −	− −	− −	− −	− −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	+ −	+ −
Raffinose	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	+ −	+ −
Inulin	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	+ −	− −	+ −	+ −	− −	+ −	+ −	+ −
Galactose	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −
Arabinose	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	+ −	+ −

+ + Acid and gas. + − Acid only. − − Negative. − + Gas only.

TABLE No. 14

GALLINARUM KLEIN

	1st Day			2d Day			4th Day			6th Day			16th Day			26th Day			44th Day		
	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract	Serum	Infusion	Extract
Dextrose	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −
Lactose	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −
Sucrose	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −
Maltose	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	− −	+ −	+ −	− −	+ −	+ −
Mannite	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	− −	+ −	+ −	− −	+ −	+ −
Dextrin	+ −	− −	− −	+ −	+ −	− −	+ −	+ −	− −	+ −	+ −	− −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −
Dulcit	+ −	− −	− −	+ −	+ −	− −	+ −	+ −	− −	+ −	+ −	− −	+ −	+ −	+ −	+ −	+ −	+ −	− −	+ −	− −
Isodulcit	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	+ −	+ −	+ −	+ −	− −
Xylose	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	+ −	+ −	− −	+ −	− −
Salicin	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	− −	+ −	− −	− −	+ −	+ −	+ −	+ −	+ −	− −
Raffinose	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	+ −	+ −	+ −	+ −	− −
Inulin	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	− −	+ −	− −	+ −	+ −	+ −	− −
Galactose	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −
Arabinose	+ −	+ −	− −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −	+ −

+ + Acid and gas. + − Acid only. − − Negative. − + Gas only.

The pullorum A strains produced gas, though there was considerable variation in the amount of gas production. Strains Number 1822 and Number 1803 never produced more than a bubble of gas during the whole period of observation, and strains Number 1791 and Number 1805 produced gas in serum water and infusion bouillon but not in extract bouillon, though Number 1805 did show a bubble of gas in the extract bouillon near the close of the period.

Lactose

All strains failed to ferment lactose.

Sucrose

All strains were negative in this sugar though in some cases a faint pink coloration in serum water was observed after a number of days of incubation.

Maltose

The sanguinarium strains showed acid formation from this sugar in all types of media after the first day of incubation. Both pullorum A and B types produced acid in serum water the first day, but were negative in the infusion and extract bouillon until about the 40th day, when they produced acid in these also. The sanguinarium strains, with the exception of "Smith," became alkaline in serum water after about the eighth day.

Mannite

All strains fermented this substance practically the same as dextrose. Gas production was variable with the different strains of pullorum A and in the different types of media.

Dextrin

All strains of sanguinarium fermented dextrin in all three types of media and, with the exception of "Smith," became alkaline in serum water after about eight days of incubation. This alkalinity was not observed in the infusion and extract bouillon. The pullorum strains produced acid in serum water from the first day if they were invigorated by transferring daily for several days in bouillon or serum water. If not previously invigorated they did not produce acid until after four or five weeks of incubation. Work which has not yet been completed indicates that they will ferment dextrin in infusion and extract bouillon also if they are repeatedly transferred into this medium containing 1 per cent dextrin.

Dulcit

The action of all strains on dulcit was practically identical with their action on dextrin. This substance was fermented by both sanguinarium and pullorum, but the sanguinarium strains fermented it somewhat more readily than the pullorum strains.

Isodulcit

All strains fermented isodulcit about equally well and there was a tendency for a return to alkalinity after an initial period of acidity, especially in serum water.

Xylose

All strains fermented xylose about equally well, though not as readily as in the case of mannite and dextrose.

Salicin

All strains failed to ferment salicin.

Raffinose

All strains fermented raffinose in serum water from the first day but were negative in infusion and extract bouillon until about the fortieth day, when acidity was noted in the sanguinarium strains, and, to a smaller extent, in the pullorum strains.

Inulin

The action on inulin of all strains was practically identical with that on raffinose.

Galactose

All strains fermented galactose readily from the first day in all types of media.

Arabinose

All strains fermented arabinose readily from the first day in all types of media.

SUMMARY OF ACTION IN SERUM WATER

The action of all strains of sanguinarium and pullorum were practically identical in serum water containing the various carbohydrates (with the exception of the gas production of the pullorum A strains). As a rule more gas was produced in serum water than any other media.

SUMMARY OF ACTION IN INFUSION BOUILLON

In infusion bouillon the action of the sanguinarium and pullorum strains was the same in all the carbohydrates except maltose, dextrin and dulcit. The sanguinarium strains fermented these carbohydrates readily while the pullorum strains fermented maltose only after about forty days' incubation and did not ferment dextrin and dulcit as long as observed. However, some of the pullorum strains fermented maltose in this medium on over-night incubation when first isolated, and all the strains fermented dextrin and dulcit after they had been kept growing vigorously in them by continued daily transfers for a number of days.

TABLE No. 15

FERMENTATION IN SERUM WATER

	PULLORUM A STRAINS					PULLORUM B STRAINS					SANGUINARIUM STRAINS			
	1791	1822	1803	1805	1454	1153	1128	1364	1150	1343	Moore	Smith	Klein	B. Sang.
Dextrose	+ +	+ +	+ +	+ +	+ +	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —
Lactose	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —
Sucrose	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —
Maltose	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —
Mannite	+ +	+ +	+ +	+ +	+ +	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —
Dextrin	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —
Dulcit	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —
Isodulcit	+ +	+ +	+ +	+ +	+ +	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —
Xylose	+ —	+ —	+ —	+ +	+ +	— —	— —	— —	— —	— —	— —	— —	— —	— —
Salicin	— —	— —	— —	— —	— —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —
Raffinose	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —
Inulin	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —
Galactose	+ +	+ +	+ +	+ +	+ +	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —
Arabinose	+ +	+ +	+ +	+ +	+ +									

+ + Acid and gas. + — Acid only. — — Negative. — + Gas only.

Table No. 16

FERMENTATION IN INFUSION BOUILLON

	Pullorum A Strains					Pullorum B Strains					Sanguinarium Strains			
	1791	1822	1803	1805	1454	1153	1128	1364	1150	1343	Moore	Smith	Klein	B. Sang.
Dextrose	+ +	+ +	+ +	+ +	+ +	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —
Lactose	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —
Sucrose	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —
Maltose	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —
Mannite	+ +	+ +	+ +	+ +	+ +	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —
Dextrin	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	+ —	+ —	+ —	+ —
Dulcit	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	+ —	+ —	+ —	+ —
Isodulcit	+ +	+ +	+ +	+ +	+ +	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —
Xylose	+ +	+ +	+ —	+ +	+ +	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —
Salicin	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —
Raffinose	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —
Inulin	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —
Galactose	+ +	+ +	+ +	+ +	+ +	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —
Arabinose	+ +	+ +	+ +	+ +	+ +	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —

+ + Acid and gas. — — Negative. + — Acid only. — + Gas only. + B Acid and bubble of gas.

TABLE No. 17

FERMENTATION IN EXTRACT BOUILLO

	PULLORUM A STRAINS					PULLORUM B STRAINS					SANGUINARIUM STRAINS			
	1791	1822	1803	1805	1454	1153	1128	1364	1150	1343	Moore	Smith	Klein	B. Sang.
Dextrose	+ —	+ B	+ B	+ B	+ +	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —
Lactose	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —
Sucrose	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —
Maltose	+ —	— —	+ —	+ —	+ —	— —	+ —	+ —	? —	— —	+ —	+ —	+ —	+ —
Mannite	+ B	+ B	+ —	+ B	+ B	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —
Dextrin	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	+ —	+ —	+ —	+ —
Dulcit	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	+ —	+ —	+ —	+ —
Isodulcit	+ +	+ +	+ B	+ —	+ B	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —
Xylose	+ +	+ +	+ —	+ B	+ B	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —
Salicin	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —
Raffinose	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —
Inulin	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —	— —
Galactose	+ B	+ B	+ B	+ —	+ B	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —
Arabinose	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —	+ —

+ + Acid and gas. — — Negative. + — Acid only. — + Gas only. + B Acid and bubble of gas.

Summary of Action in Extract Bouillon

All strains fermented the various carbohydrates in this medium in about the same manner as in the infusion bouillon, though gas production by the pullorum A strains was less marked.

Serological Proceedings

In this work two normal rabbits were immunized to each of the following strains:

1. *Bact. sanguinarium* (Moore).
2. *Bact. pullorum B*—culture No. 1343.
3. *Bact. pullorum A*—culture No. 1791.

Each rabbit was injected subcutaneously twice weekly for four weeks with the respective cultures. The antigen used for injecion was made by growing the organism on plain agar slants for twenty-four hours, washing off the growth in physiological saline solution so as to obtain a heavy suspension and then heating it for an hour at 60° C to kill the organism.

At the end of four weeks the rabbits were bled and the agglutinin titre of the serum found to be around 1-10,000. Agglutinations with the homologous antigens, cross agglutinations, and absorption tests were then carried out, as shown in the tables.

In the absorption tests each respective type of immune serum was absorbed separately with each of the three strains of organisms. The antigen used was prepared by growing on plain agar slants for twenty-four hours, washing off in physiological saline solution, and then centrifuging the organisms into the bottom of the tube and draining off the supernatant liquid. The immune serum was then added to the organisms in the tubes in the approximate proportion of three parts of a 1-10 dilution of serum in physiological saline to one part of organisms. The organisms were then thoroughly mixed with the serum and the tubes placed in the incubator at 37.5° C for three hours. They were then taken out and kept in the ice box over night. In the morning the organisms were centrifuged out and agglutinations were set using every combination of serum and antigen that it was possible to make.

Table No. 18

CROSS AGGLUTINATION

Serum	Antigen	Serum Dilutions								
		1/40	1/80	1/160	1/320	1/640	1/1280	1/2560	1/5120	1/10240
Sanguinarium	Sanguinarium	C	C	C	C	C	C	C	++	+
Sanguinarium	Pullorum B	C	C	C	C	C	C	C	++	—
Sanguinarium	Pullorum A	C	C	C	C	C	C	+++	++	—
Pullorum B	Pullorum B	C	C	C	C	C	C	C	C	+++
Pullorum B	Pullorum A	C	C	C	C	C	C	C	C	++
Pullorum B	Sanguinarium	C	C	C	C	C	C	C	++	—
Pullorum A	Pullorum A	C	C	C	C	C	C	C	C	C
Pullorum A	Pullorum B	C	C	C	C	C	C	C	C	+
Pullorum A	Sanguinarium	C	C	C	C	C	C	C	++	—

C or ++++ equals complete agglutination.

TABLE No. 19

ABSORBED SERA SET WITH SANGUINARIUM ANTIGEN

SERUM	ABSORBED BY	SERUM DILUTIONS								
		1/40	1/80	1/160	1/320	1/640	1/1280	1/2560	1/5120	1/10240
Sanguinarium		C	C	C	C	C	C	C	++	+
Pullorum B	Sanguinarium	—	—	—	—	—	—	—	—	—
Pullorum A	Sanguinarium	—	—	—	—	—	—	—	—	—
Sanguinarium	Sanguinarium	—	—	—	—	—	—	—	—	—
Pullorum B	Pullorum B	—	—	—	—	—	—	—	—	—
Sanguinarium	Pullorum B	++	+	—	—	—	—	—	—	—
Pullorum A	Pullorum B	—	—	—	—	—	—	—	—	—
Pullorum B	Pullorum A	—	—	—	—	—	—	—	—	—
Sanguinarium	Pullorum A	++	—	—	—	—	—	—	—	—
Pullorum A	Pullorum A	—	—	—	—	—	—	—	—	—

C or ++++ equals complete agglutination.

TABLE No. 20

ABSORBED SERA SET WITH PULLORUM A ANTIGEN

SERUM	ABSORBED BY	SERUM DILUTIONS								
		1/40	1/80	1/160	1/320	1/640	1/1280	1/2560	1/5120	1/10240
Pullorum A		C	C	C	C	C	C	C	C	C
Pullorum B	Sanguinarium	—	—	—	—	—	—	—	—	—
Pullorum A	Sanguinarium	—	—	—	—	—	—	—	—	—
Sanguinarium	Sanguinarium	—	—	—	—	—	—	—	—	—
Pullorum B	Pullorum B	—	—	—	—	—	—	—	—	—
Sanguinarium	Pullorum B	—	—	—	—	—	—	—	—	—
Pullorum A	Pullorum B	—	—	—	—	—	—	—	—	—
Pullorum B	Pullorum A	—	—	—	—	—	—	—	—	—
Sanguinarium	Pullorum A	—	—	—	—	—	—	—	—	—
Pullorum A	Pullorum A	—	—	—	—	—	—	—	—	—

C or ++++ equals complete agglutination.

TABLE No. 21

ABSORBED SERA SET WITH PULLORUM B ANTIGEN

SERUM	ABSORBED BY	SERUM DILUTIONS								
		1/40	1/80	1/160	1/320	1/640	1/1280	1/2560	1/5120	1/10240
Pullorum B		C	C	C	C	C	C	C	C	+++
Pullorum B	Sanguinarium	C	+	—	—	—	—	—	—	—
Pullorum A	Sanguinarium	—	—	—	—	—	—	—	—	—
Sanguinarium	Sanguinarium	—	—	—	—	—	—	—	—	—
Pullorum B	Pullorum B	—	—	—	—	—	—	—	—	—
Sanguinarium	Pullorum B	—	—	—	—	—	—	—	—	—
Pullorum A	Pullorum B	—	—	—	—	—	—	—	—	—
Pullorum B	Pullorum A	C	++	—	—	—	—	—	—	—
Sanguinarium	Pullorum A	++	—	—	—	—	—	—	—	—
Pullorum A	Pullorum A	—	—	—	—	—	—	—	—	—

C or ++++ equals complete agglutination.

Inspection of Tables 18, 19, 20 and 21 will show that:

1. In the cross agglutination tests the sanguinarium immune serum agglutinated the pullorum organisms as well as it did its own identical antigen, and vice versa.

2. In the absorption tests it was found that the sanguinarium agglutinins were practically completely absorbed by both the pullorum A and B types of organisms and vice versa, viz, both the pullorum A and B types of immune serum were completely absorbed by the sanguinarium organisms. The pullorum A and B types of organisms each completely absorbed the agglutinins specific for the other.

Summary

This work has been directed towards the differentiation of *Bact. sanguinarium* from *Bact. pullorum*. It was naturally divided into fermentative and serological studies. The results may be summarized as follows:

1. Fermentation of carbohydrates in serum water. The same carbohydrates were attacked by all strains in this medium. The pullorum A strains differed from the others by gas production. The only difference between pullorum B and sanguinarium is in the rate of fermentation of dextrin and dulcit, sanguinarium fermenting these more rapidly than pullorum.

2. Fermentation of carbohydrates in infusion and extract media. In these media the fermentations of pullorum B and sanguinarium were identical except in maltose, dextrin and dulcit. These were fermented readily by sanguinarium but with difficulty, or not at all, by pullorum. Maltose was fermented by pullorum very slowly and the other two substances were not fermented at all unless the culture had been grown in their constant presence with frequent transfers at short intervals. Pullorum A fermented the same carbohydrates as pullorum B but with gas formation from one or more of them. The gas formation was erratic.

3. Sera of rabbits immunized to representatives of one species agglutinated the other two species to the same titre as the homologous organism. This had previously been shown by Hadley(3) and Rettger(11).

4. When sera immune to each of the three types were placed in contact with organisms of the other two types practically complete absorption of the agglutinins occurred.

Discussions and Conclusions

From the data submitted in this paper it seems permissible to draw the following conclusions:

1. *Bact. pullorum*, alpha type, may be differentiated from *Bact. pullorum*, beta type, on the basis of gas formation providing a suitable nutrient basic medium is used for the tests. As has been pointed out by (Hadley(5), ordinary extract broth is not a suitable medium for this purpose. Contrary to the finding of Hadley (3) and Goldberg(2), certain strains of pullorum which were

aerogenic when freshly isolated have lost their power of gas formation after varying periods of artificial cultivation.

2. By the agglutination and agglutinin-absorption tests it is not possible to differentiate the two types of *Bact. pullorum* from each other or from *Bact. sanguinarium.* By fermentation tests it is possible to make such a differentiation between the strains studied when infusion or extract media is used as the substrate for the carbohydrates, but when serum water is used as the basic medium the characteristic fermentative differences disappear.* All of the sanguinarium strains grew much better on ordinary media than the pullorum strains. Therefore, the thought is suggested that ordinary broth is not a very favorable medium for fermentation tests with *Bact. pullorum,* and if such is the case, differences in fermentative results in this medium should not be accepted as demonstrating a specific taxonomic difference between this organism and another for which the medium was better suited.

3. This work shows that there are but few cultural differences between *Bact. pullorum* and *Bact. sanguinarium.* In fact, aside from somewhat greater growth vigor on the part of the latter, no characteristic cultural differences were found. We are not prepared to state as yet that these organisms are one and the same species, but at least this work has lent strength to the suggestion of Hadley(3) that *Bact. pullorum* may be *Bact. sanguinarium* in the making.

The writer wishes to acknowledge his indebtedness to Dr. W. A. Hagan of the Department of Bacteriology for many valuable suggestions and criticisms.

BIBLIOGRAPHY

1. BROADHURST, Jour. Infect. Dis., 1913, xiii, 404.
2. GOLDBERG, Report New York State Veterinary College, 1915–16, 283.
3. HADLEY, R. I. Expt. Sta. Bul. 174, 1918.
4. HADLEY, R. I. Expt. Sta. Bul. 172, 1917.
5. HADLEY, Jour. Bact., 1919, iv, 65.
6. JONES, Jour. Expt. Med., 1920, xxxii, 273.
7. JONES, Jour. Med. Res., 1913, xxvii, 471.
8. KLEIN, Centbl. Bakt. (etc.), 1889, v, 689.
9. MOORE, 12th and 13th Ann. Repts. U. S. Dept. Agr., 185.
10. RETTGER, Jour. Med. Res., 1908, xviii (N. S. xiii), 277.
11. RETTGER & KOSER, Jour. Med. Res., 1917, xxxv, 443.
12. SMITH & TEN BROECK, Jour. Med. Res., 1915, xxxi (N. S. xxv), 547.
13. SPRAY & DOYLE, Jour. Inf. Dis., 1921, xxviii, 43.

* This acid production might be considered by some to be due to the presence of dextrose in the serum, but when sugars were not added to the serum water there was no indication of acid production during one month of incubation.

UNDULANT FEVER IN MAN ASSOCIATED WITH BACTERIA INDISTINGUISHABLE FROM BRUCELLA ABORTUS*

VERANUS A. MOORE AND CHARLES M. CARPENTER

Department of Pathology and Bacteriology

The purposes of this paper† are to call attention to an undulant fever in man, caused apparently by infection with the organism of the Bang abortion disease, and to discuss the close relationship that exists between it and Malta fever.

For many years, a disease known as Malta fever has existed on the Island of Malta and elsewhere about the Mediterranean. It is characterized by long duration, low mortality, extremely irregular and undulating course, shifting articular rheumatism and frequent and profuse sweating. More recently it has been reported from several other countries, including the United States. In 1887, Bruce(1) discovered the cause to be a microorganism, now designated *Brucella melitensis,* but he did not explain the mode of infection. This remained in doubt until 1905 when 65 goats, all apparently in a healthy condition, were shipped from Malta to America. Their milk was drunk during the passage by the captain and many of the crew, with the result that nearly all who drank it developed Malta fever(2). On their arrival in America, *Br. melitensis* was found in the milk of several of the goats and the blood of 32 of them gave a positive agglutination test with this organism. It was reported that about half of the goats on the Island of Malta were infected with the Mediterranean (Malta) fever and that one-tenth of them were passing the organism in their milk. As a result of measures directed against drinking goat's milk there was a decided reduction in the number of cases. Sergent(3) emphasizes the possibilities of infection by contact, and the report of the commission on Mediterranean fever states that infection has taken place through small wounds.

For a long time dairy cattle have suffered from an abortion disease that appeared to be communicable. At times large percentages of the pregnant cows in certain herds have lost their calves. In 1897, Professor Bang(4), of Copenhagen, discovered a bacillus in the fetal exudate and uterine discharges which he believed to be its cause. The etiological significance of *Brucella abortus* in Bang abortion disease has been confirmed by many workers in America and Europe. A few other microorganisms have been found associated with abortion and they are believed to be the cause of the trouble in those cases. The frequency of abortions due to such organisms has not been determined, but it is apparently small.

---

* Cornell Veterinarian, April, 1926.

† Presented before the American Association of Pathologists and Bacteriologists, Albany, N. Y., April 2, 1926.

Schroeder and Cotton(5) found that nearly 14 per cent of 271 samples of milk from ten dairies contained *Br. abortus*. Smith and Fabyan(6) reported it in milk. Fabyan(7) and Schroeder and Cotton(8) found the organism in milk of cows several months after they had aborted. There is considerable literature on this phase of the subject. Zwick and Krage(9) inoculated two goats subcutaneously and one intravenously with *Br. abortus*. The organisms appeared for a period of thirty days in the milk of the one inoculated intravenously and in one of those infected subcutaneously.

Larson and Sedgwick(10) found, in 1913, in systematically testing by the complement fixation method the serum of women who had aborted, a larger number giving a positive reaction when the contagious abortion organism was used as an antigen than when the usual Wasserman test was applied. They also tested the blood of 425 children for antibodies of *Br. abortus* and found them in 72, or nearly 17 per cent. In one group of children who had been given milk from a dairy in which there had been no abortions among the cows, they did not obtain a single positive reaction. Other workers have found *Br. abortus* antibodies in the serum of children and adults.

In 1917, de Forest(11) published a series of 12 abortions in women who lived, or spent part of their gestation period, on farms where infectious abortion existed in the dairy animals. Uterine discharges from one of these cases were sent for examination to the senior author. The result was negative, but the local treatment the woman had received before the specimen was taken was believed by the sender to be the cause of the failure. The technique employed for cultivating this organism was not as satisfactory as it is today and guinea pig inoculations were not made. In his paper, de Forest refers to several practitioners who affirm that certain abortions in women were due to infection through milk. We have been unable to find any reports of human abortions associated with this organism. Carpenter, in cooperation with certain physicians, is making examinations of fetuses that are expelled without apparent cause, for the presence of *Br. abortus*. Up to the present, it has not been identified.

In 1918, Alice C. Evans(12) pointed out the very close relationship between *Brucella melitensis* and *Brucella abortus*. Feusier and Meyer(13) confirm the conclusion reached by Evans that there is only a slight serological difference between these two species of this genus. The similarity of the morbid condition in goats produced by *Br. melitensis* and that in cattle due to *Br. abortus* is in itself suggestive that *Br. abortus* may possess pathogenic properties for the human subject. Meyer, Shaw and Fleischner(14) made a careful study of the lesions produced in guinea pigs by the two organisms. They conclude that "*Br. abortus* is, as a rule, slightly more invasive and virulent than *Br. melitensis*."

A few cases of Malta fever have been reported in which the source of infection could not be traced to contact with goats, and in which no other could be established. Evans(15) has given a

review of the literature of these cases. A quotation in her paper from Kennedy(16) is significant in this connection. "I think the possibility of a melitensis infection of cows in this country should not be lightly thrust aside. I have heard of two cases of undulant fever in people who have never been out of England, and it is possible there are others undiagnosed." Khaled(17) states that he has seen cases of undulant fever in Egypt, where the patients had had no chance to ingest goat's milk and yet they suffered from typical Malta fever, as confirmed by laboratory tests.

In January, 1924, Keefer(18) reported a case of human infection with *Brucella abortus* in America. This appears to be the first case from which this organism was isolated. The clinical picture indicated Malta fever. No evidence could be found that the patient had been in contact with goats, but he had been a heavy drinker of raw cow's milk. The organism obtained from the blood was identified by Evans as *Br. abortus*. De Korte(19) reported a case of undulant fever in a man who became infected by removing the placenta from a cow that had aborted. Duncan(20) reports a case of undulant fever in a butcher who was a heavy drinker of raw milk. Orpen(21) found that a certain Rhodesian undulant fever was due to *Br. abortus*. Ficai and Alessandrini(22) report an epidemic of Malta fever in Italy. Their studies showed that certain of the cases occurred in individuals who had removed the placenta from a cow that had aborted.

In the spring of 1925, a student became ill and his case was tentatively diagnosed by his physician as typhoid fever. Later miliary tuberculosis was suspected, and a third diagnosis of malaria was suggested. Cultures were made by Carpenter from his blood and an organism indistinguishable from *Br. abortus* was obtained in pure culture. Cultures from the blood were repeated six times and always with positive results. The organism was isolated from his urine three times. The disease ran a course of about 12 weeks when recovery followed. Clinically he presented a syndrome closely allied to Malta fever. This patient had drunk heavily of raw milk which he obtained from a dairy in which abortion of a severe nature prevailed.

The second case was brought to our attention in the fall of 1925 by the same physician who attended the first case. It was that of a young man suffering from reddish, edematous spots on his legs, an undulating fever and general malaise. His blood was cultured and found to contain *Br. abortus*. He recovered in about six weeks. These two cases have been reported by the physician in charge and the junior author(23).

Through the cooperation of the physicians in charge of the Medical Division of Bellevue Hospital four other cases have been examined, from each of which this organism has been recovered. One case, a man 41 years of age, was fatal after a long illness. The autopsy showed a chronic, septic splenomegaly. The second case had three consecutive attacks, each lasting about ten days, interspersed with periods of about the same time. A third was an

atypical case of typhoid fever from which both *Br. abortus* and *B. typhosus* were recovered. We do not have a history of the fourth case, recently examined.

In the summer of 1925, the junior author made an examination of market samples of milk from the dairy from which the supply used by our first case was obtained. For a period of thirty days every sample was found, by guinea pig inoculation, to contain this organism. The herd was suffering from Bang abortion disease. Many samples of market milk from other herds, known to be affected with abortion, have been examined. They revealed the presence of the organism in a large percentage of samples.

Up to the present time we have produced abortion in pregnant heifers with each of the two strains of *Br. abortus* obtained from our first two human cases. The injection of the cultures was followed by a severe reaction and abortion occurred in twenty and six days respectively. Bacteria, identical with the ones injected, were isolated from the fetuses, placentae and milk from each case. The heifer inoculated with cultures from the first case was destroyed 6½ months later. The organism was still present in her milk, lymph glands and spleen. The cultures were very virulent for guinea pigs.

The source of human infection with *Br. abortus* has not been definitely determined. Smith(24) has studied recently strains of this organism isolated from cattle, swine and man, and reports that the evidence points to a closer relation between the human and porcine strains than between the human and bovine. Orcutt(25) found identical serological reactions with the strains with which Smith worked.

In 1914 Traum(26) isolated *Br. abortus* from the liver, kidney and stomach contents of a porcine fetus. Since that time this organism has been recovered by other observers in outbreaks of abortion in swine. The porcine strains have been reported to be more pathogenic for guinea pigs than the bovine. Schroeder and Cotton(27) produced abortion in pregnant cows by the intravenous injection of cultures obtained from swine, but not by feeding or cohabitation of swine-infected cows with other cows. Cotton(28) failed to infect by feeding pregnant sows with bovine strains. Porcine strains failed to produce abortion in two pregnant cows after intravenous injection. It is of interest to note that for three years no swine have been kept on the farms that furnished milk to our first two cases, and the men attending the cattle had no contact with hogs. We have as yet no knowledge of the possible sources of infection in the other four men, further than that they did not work in abattoirs or on dairy farms.

From our studies, it appears that milk containing certain strains of *Br. abortus* was a possible source of infection in at least two of our cases. They were both heavy consumers of raw milk. That it is possible for infection to take place through the digestive tract is evidenced by the fact that guinea pigs fed the cream and sediment of centrifuged milk from a herd affected with abortion dis-

ease developed lesions characteristic of the disease. In a previous experiment, Carpenter(29) fed milk infected with *Br. abortus* to calves and later recovered the organism from the lymph glands draining the head and neck regions.

We are continuing the study of the bacteria in the blood of cases of undulant and atypical fevers and joint troubles of doubtful etiology in man that come to us through the cooperation of local physicians and those in charge of the Second Medical Division of Bellevue Hospital in New York City. Further, we have undertaken, through the cooperation of health authorities and aid from the Heckscher Research Fund, a survey of certain market milk, not only in relation to *Br. abortus* and the pathogenesis of the various strains of the organism obtained, but also the character of its cellular contents. It is believed that such combined studies will throw additional light on the relation that appears to exist between the Bang abortion disease and certain as yet imperfectly described cases of illness in man.

REFERENCES

1. BRUCE, Practitioner, London, 1887, xxxix, 161.
2. Report of the Commission on Mediterranean Fever, 1907.
3. SERGENT, Ann. de l'Inst Pasteur, 1908, xxii, 225.
4. BANG, Ztschr. f. Thiermed., 1897, 1, 241.
5. SCHROEDER AND COTTON, 28th Annual Report, U. S. B. A. I., 1911, published 1913, p. 139.
6. SMITH AND FABYAN, Central. f. Bakt., 1912, lxi, 549.
7. FABYAN, J. Med. Research, 1912, cclxi, 441.
8. SCHROEDER AND COTTON, 28th Annual Report, U. S. B. A. I., 1911, published 1913.
9. ZWICK AND KRAGE, Berlin, tierärtz. Woch., 1913, xxix, 41.
10. LARSON AND SEDGWICK, Am. J. Dis. of Children, 1913, lxi, 326.
11. DE FOREST, Am. J. of Obst. and Dis. of Women and Children, 1917, 76.
12. EVANS, J. Infect. Dis., 1918, xxii, 580.
13. FEUSIER AND MEYER, J. Infect. Dis., 1920, xxvii, 185.
14. MEYER, SHAW AND FLEISCHNER, J. Infect. Dis., 1922, xxxi, 159.
15. EVANS, Bul. No. 143, Hygienic Lab. U. S. Public Health Service, 1925.
16. KENNEDY, J. Royal Med. Corps, London, 1914, xxii, 9.
17. KHALED, J. Hyg., Cambridge, Eng., 1921, xx, 319; Ibid., 1924, xxii, 335.
18. KEEFER, Bul. Johns Hopkins Hosp., 1924, xxxv, 6.
19. DE KORTE, S. African Med. Record, 1924, xxii, 478.
20. DUNCAN, British Med. J., 1925, No. 3352, 554.
21. ORPEN, S. African Med. Record, 1924, ccxxi, 75.
22. FICAI AND ALLESSANDRINI, Il. Policlinico Seg. Post., Rome, 1925, xxxii, 113.
23. CARPENTER AND MERRIAM, J. Am. Med. Assoc., 1926, lxxxvii, 1269.
24. SMITH, J. Exp. Med., 1926, xliii, 207.
25. ORCUTT, Ibid., 1926, xliii, 225.
26. TRAUM, quoted by Hayes, J. Am. Vet. Med. Assoc., 1924, lxv, 211.
27. SCHROEDER AND COTTON, J. Am. Vet. Med. Assoc., 1925, lvi, 550.
28. COTTON, J. Am. Vet. Med. Assoc, 1922, lxii, 179.
29. CARPENTER, Cornell Vet., 1924, xv, 16.

AGGLUTININS FOR BRUCELLA ABORTUS IN THE BLOOD SERUM OF MAN*

C. M. CARPENTER

DIAGNOSTIC LABORATORY

Department of Pathology and Bacteriology

Much evidence has been presented recently to show that certain strains of *Brucella abortus* may produce disease in man. For the most part the diagnosis of these infections has been accomplished by isolating the organism from the blood of these patients and by finding the specific agglutinin in the blood serum. The agglutination test for infectious abortion in cattle has been used extensively since its value was demonstrated by Grinsted(1) and Holth(2). It is not known just what percentage of infected animals does not show *abortus* agglutinins, but we are certain it is low.

Larson and Sedgwick (3) observed that in complement fixation tests of the sera of women that had aborted, a greater per cent showed the presence of *abortus* antibodies than gave a positive Wasserman reaction. In studying the blood serum of 425 children they found, by the agglutinin absorption test, specific *abortus* agglutinins in 17 per cent. They showed that children who had drunk milk from a herd where infectious abortion did not exist, did not harbor any of these agglutinins. Nicoll and Pratt(4), Cooledge(5), and others, have shown that specific *abortus* agglutinins were present in the serum of persons who had drunk raw milk containing *Brucella abortus*, while Nicolle, Burnett and Conseil(6) have demonstrated their presence in the serum of men injected with this organism. Evans(7) examined the blood serum of 500 patients suffering from various diseases and found that fifty-nine contained specific agglutinins for *Brucella abortus*.

Keefer(8) reported the first case of *Brucella abortus* infection in America. The titre of the serum from this case was 1:3200. Evan(7) reported finding *abortus* agglutinins in three cases, one of which showed a titre of 1:160, while the other two had a titre of 1:320. Duncan(9) reported an *abortus* agglutinin titre of 1:2400 in a patient suffering from undulant fever. Orpen(10), DeKorte(11), Ficai and Alessandrini(12) and Viviani(13) have reported similar cases which were undoubtedly due to *Br. abortus*. In these cases evidence pointed to infection from cattle. The blood serum from these patients had a fairly high titre.

During the last year I have studied twenty cases of undiagnosed fevers. (See Chart I.) Cultures were made and guinea pigs injected with the blood and, in many instances, with the urine, from each of the patients. Agglutination tests with the *abortus* antigen were made and with those sera where agglutination occurred, with the exception of Case 15, agglutinin absorption tests were made to determine their specificity.

* I have had financial aid from the Heckscher Fund for Research.

Brucella abortus was recovered in culture from five of these cases, but agglutinins for this organism were present in only three of the five. Specific *abortus* agglutinins were present in blood serum of Case 13, but *Brucella abortus* was never recovered in culture from the blood. However, cultures for *abortus* were not made until after the symptoms had subsided. The agglutinins did not appear in the blood serum until the end of the disease course which lasted approximately two months. Although the *abortus* antigen was agglutinated by the serum from Case 15, we did not have enough to complete an agglutinin absorption test. Cases 1 and 7, from which *Br. abortus* was isolated in pure culture, showed typical symptoms of undulant fever. Case 4, which was diagnosed by the clinician as subacute bacterial endocarditis, gave a culture of *Br. abortus*. This case was fatal and autopsy revealed an acute nephritis and marked splenomegaly.

Cases 8 and 16 are extremely interesting because *abortus* agglutinins were not present even in dilutions of 1 : 5. In Case 8 *Bacterium typhosus* was recovered in culture with *abortus* and the serum gave a positive Widal when it was diluted 1 : 60. The serum of the latter showed a four plus Wasserman and because of this and a general lymphadenitis, a diagnosis of syphilis of the lymph nodes was made by the clinicians in charge of the case. A lymph node was removed from Case 16 and sectioned. The examination revealed foci of infection, the description of which was similar to that of those lesions observed in lymph glands of guinea pigs injected with *abortus*. The slides were not seen by the writer. It is difficult to interpret these findings and to know what significance should be given *Br. abortus* as a factor in producing disease in these two cases. At the onset of the disease in Case 8, undulant fever was suspected by the physician in charge because of profuse night sweats, a negative Widal and absence of rose spots. Later the course of the disease became more typical of typhoid and a satisfactory recovery was made. It is possible that the typhoid infection had produced an inflammation, and perhaps ulceration, of the intestine, facilitating the invasion of the blood by *Brucella abortus*.

It is not known whether *abortus* agglutinins are produced in the presence of a positive Wasserman. Many observers conclude that in multiple infections and immunization, the production at the same time of different agglutinins takes place without any change in the onset, intensity and duration. Hektoen(14), on the other hand, believes that the normal development of agglutinins is altered in secondary infections. Lewis and Loomis(15) have shown that the allergic irritability is increased in guinea pigs in the presence of tuberculosis. Schoenfeld and Carpenter(16) showed that guinea pigs suffering from tuberculosis readily developed *abortus* agglutinins when they were infected subsequently with the latter organism.

The two cultures isolated from Cases 8 and 16 produced abortion in susceptible pregnant heifers and appeared to be as virulent for

them, and for guinea pigs, as were the other three strains which had developed specific agglutinins in the blood serum of the patients from which they were isolated.

It has been observed frequently that the blood serum of an aborting cow will not show the presence of *abortus* agglutinins until a few weeks after the expulsion of the fetus. The placenta may show typical lesions of the disease and a culture of the organism can be recovered from it as well as from the tissues of the fetus.

A review of the literature on the agglutination test for diagnosing Malta fever shows that the serum from a large percentage of cases had a titre of from 1:10 to 1:100. There is a great difference of opinion among the various authors as to the titre of a serum that should be considered as conclusive evidence of Malta fever. Basset-Smith(17) favors a positive reaction in a dilution of 1:30, although he states that some cases of Malta fever occur where no agglutinins for *Br. melitensis* can be demonstrated. Other workers have had this same experience.

Inasmuch as *Brucella abortus* is at present considered only a variety of *Brucella melitensis*, it is to be expected that those strains which have been recovered from man will stimulate the production of agglutinins in the patient's serum in a manner and quantity not unlike *melitensis*. It is well known that certain strains of *Brucella melitensis* and *Br. abortus* make superior antigens while many others are unsatisfactory. This factor in itself may be responsible for variation occurring in the serum titre of many cases reported. We have used as an antigen in this work a living suspension of *Brucella abortus*, "80," in physiological salt solution. The antigen gave a reading of 2.5 cm. on the Gates(18) apparatus for determining the density of suspensions. The readings were made after twelve hours' incubation. The technic of the agglutinin absorption tests is practically identical with that employed by Evans(7). A heat-killed antigen was used with a density of approximately 48,000 p.p. M. after the serum, which had been diluted 1:10, was added.

SUMMARY

The serum from four out of twenty cases, showing symptoms suggestive of undulant fever, showed specific agglutinins for *Brucella abortus*. The serum from a fifth case which showed typical symptoms of recurrent or undulant fever, agglutinated the *abortus* antigen completely in a dilution of 1:120. We were unable to obtain sufficient serum for an agglutinin absorption test.

Brucella abortus was isolated from the blood of five of the twenty cases. In two of these no *abortus* agglutinins could be demonstrated in the blood serum. However, one gave a positive Widal while the other showed a four plus Wasserman.

The titre of the sera varied from 1:30 to 1:480.

In the light of these findings it is evident that the agglutination test cannot be relied upon to detect the presence of *Br. abortus* in man.

I am grateful to Dr. Alice Evans of the United States Hygienic Laboratory for assistance with the agglutinin absorption tests and to Dr. George J. Young of New York City for collecting much of the material examined. Thanks are also due to many others who have cooperated with me in this work.

CHART I

RESULTS OF AGGLUTINATION TEST ON BLOOD SERUM OF 20 PATIENTS SHOWING SYMPTOMS RESEMBLING THOSE OF UNDULANT FEVER

No. of case	Diagnosis	Bacteriological Findings	Agglutination Test with *Brucella abortus* Antigen							
			Serum diluted to							
			5	15	30	60	120	240	480	960
1	Undulant fever	Br. abortus	+	+	+	+	+	+	+	—
2	Not made	Staph. albus	—	—	—	—	—	—	—	—
3	Not made	Negative	—	—	—	—	—	—	—	—
4	Subacute bacterial endocarditis	Br. abortus	+	+	+	+	S	—	—	—
5	Not made	Negative	—	—	—	—	—	—	—	—
6	Not made	Negative	—	—	—	—	—	—	—	—
7	Undulant fever	Br. abortus	+	+	+	—	—	—	—	—
8	Typhoid fever	Br. abortus Bact. typhosus	—	—	—	—	—	—	—	—
9	Typhoid fever	Bact. typhosus	—	—	—	—	—	—	—	—
10	Typhoid fever	Negative	—	—	—	—	—	—	—	—
11	Not made	Negative	—	—	—	—	—	—	—	—
12	Subacute bacterial endocarditis	Streptococcus	—	—	—	—	—	—	—	—
13	Not made	Negative	+	+	+	—	—	—	—	—
14	Not made	Negative	—	—	—	—	—	—	—	—
15	Undulant fever	Unidentified G-rod	+	+	+	+	+	S	—	—
16	Syphilis of lymph nodes	Br. abortus	—	—	—	—	—	—	—	—
17	Influenza	Negative	—	—	—	—	—	—	—	—
18	Not made	Negative	—	—	—	—	—	—	—	—
19	Acute articular rheumatism	Negative	—	—	—	—	—	—	—	—
20	La grippe	Negative	—	—	—	—	—	—	—	—

+ Indicates complete agglutination.
S Indicates slight agglutination.
— Indicates no agglutination.

BIBLIOGRAPHY

1. GRINSTED, P., Maandsskr. fr. Drylaege., 1909, 21, p. 395.
2. HOLTH, H., Berl. tierärtz. Woch., 1909, 25, p. 686.
3. LARSON AND SEDGWICK, Am. J. Dis. Child., 1913, 6, p. 326.
4. NICOLL AND PRATT, Am. J. Dis. Child., 1915, 10, p. 203.
5. COOLEDGE, L. H., J. Med. Res., 1916, 34, p. 459.
6. NICOLLE, BURNETT AND CONSEIL, Compt. rend. Acad. des Sci., 1923, 176, p. 1034.
7. EVANS, A. C., Bull. 143, U. S. Hygienic Lab., 1925.
8. KEEFER, C. A., Bull. Johns Hopkins Hosp., 1924, 35, p. 6.
9. DUNCAN, J. T., Brit. Med. J., 1925, No. 3351, p. 554.
10. ORPEN, L. J., J. South African Med. Rec., 1924, 22, p. 75.
11. DE KORTE, W. E., South African Med. Rec., 1924, 22, p. 478.
12. FICAI AND ALESSANDRINI, Il Policlinico, 1925, 32, p. 113.
13. VIVIANI, R., Il Policlinico, 1925, 32, p. 203.
14. HEKTOEN, L., Harvey Lectures, 1909–1910, p. 163.
15. LEWIS AND LOOMIS, J. Exp. Med., 1924, 40, p. 503.
16. SCHOENFELD AND CARPENTER, J. Inf. Dis., 1925, 37, p. 68.
17. BASSETT-SMITH, P. W., Rep. on Med. Fever, Roy. Soc. London, 1906, Pt. 4, p. 101.
18. GATES, F. L., J. Exp. Med., 1920, 31, p. 105.

A COMPARISON OF STRAINS OF BRUCELLA ABORTUS ISOLATED FROM MAN WITH THOSE FROM CATTLE*

C. M. CARPENTER

DIAGNOSTIC LABORATORY

Department of Pathology and Bacteriology

Recent literature has contained reports of cases of undulant fever in man from which cultures of an organism indistinguishable from *Brucella abortus* have been recovered. This has prompted investigators to discover, if possible, the source of this infection. In 1918 Evans(1) reported that *Brucella abortus* and *Brucella melitensis* are very closely related in all of their biological characteristics and that *Brucella abortus* is only a variety of *Brucella melitensis*. She has found, however, that by means of the agglutinin absorption test the two strains may be differentiated with certainty.

During the last year the author has had the opportunity to study five strains of *Br. abortus* that he has isolated from the blood and urine of man and to compare them with strains of *Brucella abortus* recovered from bovine fetuses, placentae and milk. They have been examined with reference to their atmospheric requirements, as evidenced by their original isolations, their serological relationships, their pathogenicity for guinea pigs, their effect upon pregnant cattle when injected intravenously, and their ability to establish themselves in the udders of cattle.

When Bang(2) first described *Brucella abortus* as the cause of infectious abortion in cattle, he stated that it had anerobic tendencies and grew better in an atmosphere where the amount of oxygen was reduced. Still further work has shown that it is not a reduction in oxygen that facilitates its growth but rather an increased amount of carbon dioxide. Smith and Fabyan(3) and Schroeder and Cotton(4) observed that the Bang organism was pathogenic for guinea pigs. It produced a chronic progressive disease similar to tuberculosis, except that the majority of the guinea pigs did not lose weight or die and that some pigs evidently recovered from the infection.

Although *Brucella abortus* occurs more frequently in cattle than in any other species of domestic animal, it has been recovered from sheep, horses and swine. Quite extensive outbreaks of abortion in swine due to *Brucella abortus* have been reported. The porcine strains that have been isolated are reported to grow readily upon nutrient agar without an increased amount of CO_2 or sealing of the tube. They are also considered to be more pathogenic for guinea pigs than the bovine strains. According to Smith(5) the primary lesions produced in the tissues of guinea pigs by both strains are identical, but as the disease progresses the porcine strain produces a lesion which shows a more extensive infiltration with

* Presented before the 26th Annual Meeting of the American Association of Pathologists and Bacteriologists, April 2, 1926, Albany, N. Y.

polymorphonuclear leucocytes often followed by a central necrosis. Nearly all workers have produced abortion in cattle by injecting the bovine strain intravenously into pregnant cattle, but abortion has not been produced regularly in cattle by injecting porcine strains intravenously.

One of the difficulties encountered in studying the comparative virulence of various strains of *Brucella abortus* is a decreased pathogenicity of the organism for guinea pigs after it has been cultivated on culture media for various periods of time. Huddleson(6), as well as many other observers, has reported strains that at one time produced typical lesions of *Br. abortus* infection in guinea pigs while perhaps a year later the same strain was nonvirulent for guinea pigs. Porcine, as well as bovine, types have shown this same tendency. Whether this is true of the strains isolated from man, cannot be stated at this time. The biological characteristics of five of these strains have been studied.

The recovery of the organism from man was made by guinea pig injection and by cultures from the blood and urine on nutrient agar, to which sterile horse serum had been added. The cultures were made by adding to the media from 1 to 3 cc. of blood from the patient. The tubes were sealed with sealing-wax and incubated at 37°C. Four of the strains were recovered in this manner while the fifth was grown in an atmosphere where 15 per cent of the air had been replaced by 10 per cent. of CO_2. The first generation of these strains has always been difficult to grow when the culture tubes are sealed with wax. In the majority of instances no growth has developed for 6 or 8 days. It has also been necessary to open the tubes once or twice and smear the mixture of blood and condensation water over the slanted surface of agar to obtain an observable growth. The second and following generations, however, have grown well on plain agar without sealing the tubes, although better growths have been obtained by sealing. The last strain which was isolated in an atmosphere of increased CO_2 showed a good growth in 48 hours.

The positive identity of the organisms isolated was established by the agglutinin absorption test. The various strains were checked against known strains of *Brucella abortus* and anti-abortus sera. The cultures isolated from man could not be differentiated by serological methods from those recovered from cattle.

The first human strain which was isolated has proved to be slightly more virulent for guinea pigs than the other four and has developed lesions similar to those described by men who have worked with the porcine strains. The other four are more virulent for guinea pigs than the usual bovine type, but I have worked with several bovine strains that have shown just as marked virulence. As a rule the guinea pigs injected with from 1 to 3 cc. of blood from the patients or with a small amount of the culture isolated from the blood, lost weight, rather than increased in weight as is customary with most of the bovine types. In several instances they became markedly emaciated. Cultures of *Br. abortus* extremely

virulent for guinea pigs were isolated daily from the milk drunk by the patient mentioned in the first case. Examination of the milk from the individual cows comprising the herd revealed that 50 per cent of the animals harbored *Brucella abortus* in their udders and that the milk from two of the cows was heavily charged with strains of *Brucella abortus* more pathogenic for guinea pigs than were the strains isolated from the other animals. Many of the guinea pigs that were injected with the sediment and cream of mixed milk from this herd became greatly emaciated and died from fourteen to forty days after the injection. They showed extensive lesions of *Brucella abortus* infection and the epididymes and testes were invariably abscessed. The seminal vesicles were filled with pus in many cases. I have observed also extremely virulent bovine strains of *Brucella abortus* in other instances. I would like to mention one in particular isolated by K. F. Meyer(7) from certified milk. This culture was labelled by him "80" and in 1923 small amounts of this culture would kill guinea pigs in three or four weeks. There were extensive lesions in the joints, reproductive organs, spleens and livers. Abscesses were present in the omentum and lymph glands. One of my colleagues who has been working recently with this same culture has told me that it still shows marked virulence for guinea pigs.

The intravenous injection of each of these human strains of *Brucella abortus* into pregnant heifers has been made. Five heifers in their first pregnancies were obtained from herds where no abortion had existed. Their blood serum was examined several times by the agglutination test for infectious abortion and found negative before they were injected. Ten mils of washed agar cultures suspended in physiological salt solution were injected. Three of the suspensions gave a reading of 2 by the Gates(8) method of determining the density of suspensions, while the other two were slightly heavier and gave a reading of 1½. These five heifers aborted from four to twenty days after receiving the cultures. The organism has been recovered from the placenta and milk in each case. Four of the fetuses gave pure cultures of the organism injected, but the fifth fetus was expelled alive at approximately the eighth month of pregnancy. Inasmuch as we needed calves to nurse the dams he was not destroyed and cultured. Two of the cows became moribund from a pyometra and pyemia one month after abortion. They were destroyed and autopsied. Cultures of *Br. abortus* were obtained from the abscesses and uterus of each.

The injection of the same amount of *Br. abortus* from bovine sources by Birch and Gilman(9), as well as other workers, has not produced abortion in such short periods of time as has that of cultures isolated from man. Neither have they produced in the experimental animals as marked a reaction as have the human strains. The bovine types do not always establish infection in the udder when cultures of the organism are injected intravenously. A significant fact in connection with these experiments is that in one cow the mammary gland became infected and *Br. abortus* was

discharged continuously for 231 days, when she was destroyed. A calf which had nursed this cow became infected and, although it did not have any milk for four months after the dam was removed, it was still infected as evidenced by a high agglutinin titre and by the presence of *Br. abortus* in many of the lymph glands. In the earlier work(10) on this problem of calf infection from milk it was shown that as a rule *Br. abortus* cannot be recovered from the calf six weeks after the infected milk is removed.

CONCLUSIONS

1. The first generation of cultures of *Brucella abortus* isolated from man was difficult to cultivate, but the second and following generations have grown well on unsealed tubes of nutrient agar.
2. The human strains have proven very virulent for guinea pigs, but not more virulent than certain bovine strains with which the author has worked. Four of the strains have produced lesions in guinea pigs which could not be distinguished from lesions produced by virulent types isolated from milk, placentae and bovine fetuses.
3. Abortion was produced in five pregnant heifers by the intravenous injection respectively of five of the cultures. The organism was recovered from the colostral milk, placentae and fetuses. The infection established itself in the udder and was present in the milk of one case for 231 days after the animal aborted. The cultures isolated from man were more toxic for the pregnant heifers than the bovine types and produced abortion in a shorter time than have bovine strains as reported by other observers.

I am indebted to Alice C. Evans of the U. S. Hygienic Laboratory for assistance in the serological work and in identification of the cultures. I have also had financial aid from the Heckscher Fund for Research.

BIBLIOGRAPHY

1. EVANS, ALICE C., J. Inf. Dis., 1918, 22, 580.
2. BANG, B., Ztschr. f. Thiermed., 1897, 1, 241.
3. SMITH AND FABYAN, Cent. f. Bakt., Parasit. und Infekt., 1912, 61, 549.
4. SCHROEDER AND COTTON, 28th Annual Report, U. S. B. A. I., 1911, 139.
5. SMITH, T., J. Exp. Med., 1926, 43, 207.
6. HUDDLESON, I. F., Technical Bulletin 55, Agr. Exp. Sta., Mich. Agr. College, 1922.
7. MEYER, SHAW AND FLEISCHNER, J. Inf. Dis., 1922, 31, 159.
8. GATES, F. L., J. Exp. Med., 1920, 31, 105.
9. BIRCH AND GILMAN, Annual Report New York State Vet. College at Cornell University, 1922–23, 62.
10. CARPENTER, C. M., Cornell Veterinarian, 1924, 14, 16.

GROWTH OF VETERINARY SCIENCE AND THE APPLICATION OF NEW KNOWLEDGE*

VERANUS A. MOORE, *Dean*

"The whole of medicine has been established since long ago; and a principle and a way has been found to discover, as has already been done, many excellent things, which, in turn, shall serve to discover a vast number of others, provided that he who makes search is fitted for it, and that, having knowledge of what has been found, he follows the same trail."—*Hippocrates.*

The growth of veterinary medicine started at the time when those who practiced the art recognized and applied the teachings of science. So long as practice was guided by opinions and superstitions, progress was slow but when definite facts and laws pertaining to disease were revealed through the biological and physical sciences and the new knowledge was applied in diagnosis and treatment, veterinary medicine became an applied science. Its growth has been measured by the new discoveries that have been made and the extent to which they have been utilized. In recent years facilities for research have been increased and consequently new knowledge pertaining to animal diseases has been accumulated more rapidly than it has been assimilated in the routine of the profession, which means that there is available considerable technical information as yet not utilized by practitioners.

We are living in an age of great progress, "the most prodigious period of change through which the world has ever passed." The succession of discoveries in veterinary sciences is augmenting and changing our knowledge of disease and its control with such rapidity that practitioners and laboratory workers are forced to revise their theories, interpretations and methods quite as frequently as manufacturers have to replace their equipment for that of more modern design. The composite of knowledge that dominates the instruction in a curriculum for the definite time during which a student is an undergraduate, like an ocean wave, soon breaks on the beach and returns in part as an undertow; and unless we are receptive to the new information that succeeding researches reveal, we soon become a part of the undercurrent that is drawing the profession backward rather than forward. The key note of progress is to discard that which is outworn, cling to that which is true, and welcome new methods that enable one to increase his ability to meet the ever-growing demands in the service he is trying to render.

An influence that is retarding progress in veterinary medicine is the imaginary chasm between its science and its practice. Scientists evolved the theory and demonstrated the fact that diseases

---

* Presented at the Conference for Veterinarians, N. Y. State Vet. College, Jan. 14, 1926.— *Cornell Veterinarian,* April, 1926.

are due to natural causes and the acceptance of the theory called at once for the application of scientific methods with which to combat them. It was difficult for those who entered practice without adequate experience in scientific work to understand and apply the new knowledge, for it was often contrary to the accepted theory and practice with which they were familiar. It required a generation to establish the principle of a specific etiology and many years more to formulate, through experimentation, the accepted principles underlying immunity. Fortunately, we are now quite removed from that situation as most practitioners of today have had laboratory training which should qualify them to utilize recent findings.

We must not be unmindful of the tendency in the medical professions to go to extremes when new discoveries of much promise are reported, and to substitute the new procedure for that which previously had been employed and found reasonably safe and efficient. Laboratory methods do not supplant physical examinations and the syndrome of disease in diagnosis, but they are valuable adjuncts to them. Often they are the only means of making a definite determination possible. A diagnosis of anthrax from the history and symptoms might be uncertain, but if the examination of the blood showed *Bacterium anthracis*, the doubt would be removed. If the examinations were negative the syndrome would remain the chief reliance for the identification of the malady. A positive reaction following the use of tuberculin is considered evidence of the presence of tuberculosis, but a failure to obtain a reaction, in an animal with physical evidence of the disease, should not be considered as a guarantee that the individual is free from it. It is important in the diagnosis and control of disease that every aid experience and science have given us should be utilized. If to remove a doubt it is necessary to resort to microscopical examinations, bacteriological methods or specific tests, they become integral parts of the diagnostic procedure quite as much as taking the temperature or counting the pulse. Methods of precision have their origin in pure science, but their real value lies in the assistance they give the practitioner in determining the nature of the disease in question. All information that can be brought to bear on the diagnosis or control of a disease should be fused into a carefully systematized and more accurate procedure. There is, therefore, no dividing line between the scientific knowledge of a disease and that which should be applied in its diagnosis and treatment.

There are many illustrations of the immediate benefits that have followed the practical application of the findings of research in the handling of disease. The use of mallein in diagnosing glanders and tuberculin in detecting tuberculosis are aids that researches have given the clinicians. Hog cholera, that formerly was a serious scourge, can be kept under complete subjection by following the teachings of science relative to its etiology and con-

trol. The prevention of losses due to malnutrition, made possible by the newer conception of the significance of vitamins and salts in food, is another example of the value of scientific research in extending the usefulness of professional service. A veterinarian told me recently that in one case the losses in poultry reported to him as either fowl plague, fowl typhoid or chicken cholera, were found on investigation to be due to improper feeding and parasites. This increment of definite information, added to his former knowledge and experience, enabled him to remove a condition that loomed before the poultrymen as an uncontrollable situation. Further examples are not necessary to bring into bold relief the progress veterinary medicine has made, because of the application of scientifically determined facts and more careful and accurate technique.

In reviewing the accomplishments already recorded, one is amazed at the success in overcoming the natural forces that tend to destroy animal life. However, the many unsolved problems in live stock sanitary work and the control of spreading destructive diseases remind us that we have just begun to acquire and apply knowledge that is needed to insure the maximum protection against losses of this kind. It is for that reason that new findings are welcome. Many of those recently recorded are interesting and may prove to be valuable, but I desire to call your attention especially to the situation created in very recent time by the development of a means of identifying infected individuals in two very important diseases. I refer to the agglutination test as a diagnostic agent in *bacillary white diarrhea* of chickens and the *Bang abortion disease* in cattle.

In the past the acquisition of new knowledge and its application has extended the usefulness of veterinarians by bringing one disease after another under control. Today bacillary white diarrhea in chickens and the Bang abortion disease in cattle stand out among the serious, if not the most destructive, diseases that threaten the poultry and dairy industries of the country. They have been the occasion of much study and research not only in this college, but in many other institutions. Finally, we have arrived at a stage of knowledge concerning them where we have a method, or test, of great value in identifying infected individuals, which is the first and fundamental step in the control of these maladies. Men interested in poultry and dairy husbandry are clamoring for assistance, and now there are methods available whereby veterinary practitioners can render this longed-for and important service.

The utilization of the antibody known as agglutinin was first applied as a diagnostic method for typhoid fever. You are familiar with it under the title of the "Widal test." In veterinary medicine, it was used by Schütz and Miessner for the diagnosis of glanders, especially in obscure cases. Recently, it has been applied quite extensively for detecting fowls harboring *Bacterium pullorum* and cattle infected with *Brucella abortus*. The technique of the

test and its application as a diagnostic agent in these infections have been studied carefully by many workers. The literature on the test is voluminous and the majority of conclusions testify to its efficiency.

The diagnosis by means of absorbing the complement in the blood and known as the "complement fixation method" is believed by many workers to be equally as efficient as the agglutination test. It is, however, a much more complicated procedure and consequently cannot be applied advantageously when large numbers of examinations are to be made.

The disease known as *bacillary white diarrhea* spread rapidly with the development of the poultry industry until it has become the cause of very heavy losses, especially burdensome to those who deal in day-old chicks. Its study revealed the fact that infected chicks that recover often harbor in their adult life *Bacterium pullorum* in their ovaries. The eggs from such hens were found frequently to be infected with the organism and chicks hatched from such eggs became victims of the disease and a means of spreading it to those in contact with them. It was soon realized that the control of this malady centered in finding the laying hens that were carriers of the organism and excluding them from the flock whose eggs were used for hatching. The finding of the specific agglutinin in the blood of such fowls pointed to the agglutination test as a means of detecting the offending individuals.

Numerous criticisms have been made relative to the efficiency of this test. Here again has been displayed a disregard for the way nature has provided for the dissemination of this organism. We are told that healthy chicks have been hatched from the eggs of hens that react to the test. An examination of the reports by Jones from our laboratory, Gage from Amherst, Mass., and others, will show that many of the eggs from such hens are not infected and it is not impossible that a small percentage of reacting fowls may lay uninfected eggs throughout their lives. These records show, however, that some of the eggs from most reacting fowls are infected, and that the agglutination test will detect most of the carriers of the organism. No thoughtful man, experienced in these things, is bold enough to believe that this, or any other method, is 100 per cent efficient. While much additional work is necessary, the findings have been so encouraging that many poultrymen desire to have certified flocks from which eggs, or chicks, could be sold with a guarantee against losses from bacillary white diarrhea. This calls for the application of this test. As with tuberculin, many laymen do not understand the limitations of specific tests and when failure occurs they blame the method. They need the services of veterinarians to explain the difficulties, answer their questions and guide them in the control work.

There are many facts to be ascertained relative to the control of bacillary white diarrhea, such, for example, as the communicability of the infection from adult carriers to uninfected fowls and the

number of tests that are necessary to detect all of the "carriers" in a flock. These will be determined through experimentation and the observations of those working professionally with the malady. Many hatcheries are anxious to insist on the incubation of eggs from hens free from this infection. As the agglutination test is believed to be the most reliable method for the identification of adult "carriers," it is quite natural that the demand for it is increasing. Last winter poultrymen appealed to the Legislature for assistance in eradicating this scourge from their flocks. This appeal is bound to become more and more insistent unless relief is obtained through the veterinarians.

The value of the agglutination test in the detection of cattle infected with *Brucella abortus* is becoming clear. The test is specific for this infection and for no other. Those who have used it consistently in practice testify to the help it affords in differentiating the Bang abortion disease from other infections and in directing the management of the herds. While its interpretation involves many factors, practically all who have used the test agree that it has an important place in the control of Bang abortion disease. Several States require a negative agglutination test on all cattle allowed to enter. In one State conferences are being held this month for the purpose of seeking legislation calling for free tests. This suggests that ultimately there will be state supervision and effort to control this malady unless veterinarians interest themselves in it. Abortion is causing heavy losses and owners are anxious to obtain relief from it. They reason that if the state can test and indemnify for tuberculosis it can for this disease. We believe that infectious abortion of whatever cause can be controlled far better by local veterinarians than by any central or remote agency.

The questions that confront the veterinary profession today relative to these two diseases are very definite and clear. Poultrymen and cattle owners are deeply interested in these maladies and many of them are anxious to eliminate the loss caused by their presence. They are asking, in ever-increasing numbers, for the test. For a number of years, while it was being developed, we made the test free of charge as a research problem. Now that it is given the distinction of being a specific diagnostic measure it belongs to the clinicians. It is needed by them to recognize the presence of the infection quite as much as the thermometer is required to determine the degree of temperature. The time has come when, because of the number of owners asking for the test, different arrangements must be made. To discourage the curious and to aid the earnest we are making, on a fee basis, as many tests for *B. abortus* as we can, the fee being used for additional help. We are continuing to study the test in *bacillary white diarrhea* by making it for a few poultrymen who cooperate with us by reporting the extent of the disease that appears in the chicks hatched from their flocks. This, however, does not satisfy the demand. Poultrymen believe they

should make the test and remove the reactors from the flock from which eggs for hatching are obtained. They apply to us for assistance and we in turn refer them to their veterinarians. I am sorry to say, the reply comes back too often that their veterinarian says he is not interested in poultry diseases. Occasionally a similar statement comes in reference to abortion in cattle.

The agglutination test for these two diseases stands in exactly the same relation to practice that tuberculin did twenty years ago. Local practitioners, as a rule, did not interest themselves in teaching their clients how to detect and remove tuberculous animals from their herds. They often were careless and indifferent to the technical requirements for its eradication, in the use of tuberculin, and in reporting accurately and promptly the results of the test. As a consequence, its use was taken temporarily from them and given to State veterinarians only. Shall the agglutination test for the diseases in question follow the same course? Is it not the purpose of practicing veterinarians to diagnose, prevent, treat and control animal diseases? Are they not the logical leaders and teachers in all live stock sanitary work? Is it not better for the State and the morale of the public that people who receive special benefit for their personal gain should pay for the service? The conditions under which these infections exist are so varied that the handling of each flock or herd must be guided according to the principles involved rather than by a uniform, prescribed method. Further, the owners must be instructed how to protect their animals. The practitioners are the advisers to whom they can go for answers to the multitude of questions that necessarily arise in the symptom-complex of disease control.

Three ways have been suggested by which the agglutination test can be made available, namely:

(1) *By practitioners.* There are young veterinarians enough, scattered throughout the State, who have had laboratory training, and who, I believe, are interested in this work, who can make this simple, but technical, test. The equipment, if the antigens are obtained from the college, or elsewhere, is inexpensive. If local men do the work it would provide advisers on the subject within reach of all owners. It would increase interest in veterinary medicine, dignify the profession and add materially to the income of those who do the work. In other words, the testing could be done by men in the field on a fee basis to the advantage of every one. I do not mean that every practitioner should make the tests, for all are not qualified; but I do believe that a large enough number of them have had adequate laboratory training as a background of preparation to qualify in the details necessary to make this a practitioner's specialty. We have specialists in surgery; in small animal diseases; and in breeding troubles. Why not have special-

ists in the simpler laboratory methods of diagnosis for which there is a growing demand? In conjunction with the actual tests which the owners wish made, many questions arise in their minds that should be answered. The veterinarians who are doing the work are naturally the best counselors, for they know the local conditions.

(2) *At a central laboratory on a fee basis.* The State could build and equip a laboratory here, or elsewhere, large enough for this work, and do the testing on a fee basis. In this way it would secure funds to pay technicians to do the work and provide necessary supplies.

(3) *Free laboratory service.* There are those who advocate that the State should do this work entirely, and free of charge. They imply State supervision and control. I fail to see why the expense of this service should be placed on the taxpayers. Further, it would centralize the work, which would deprive owners of technical advisers within reach, whom they need to consult relative to the application of the new knowledge in their particular circumstances. If these diseases are to be eliminated, and the flocks and herds kept free thereafter, the owners must be instructed generally in the nature of the maladies, the procedures necessary to remove the infection and the precautions to be taken afterwards. The practitioners who are in personal touch with the owners are their logical teachers and counselors along these lines.

The economic importance to the animal husbandry of the State of having competent veterinarians within reach emphasizes the desirability of having practitioners familiar with all questions bearing on the disease of animals in their community. For that reason the solution of the problems before us seems to be that practitioners enough equip themselves for the work and do it. The college will assist gladly those who wish special instruction in any of the details and it will furnish the antigens.

The function of the college should be understood clearly. In addition to teaching undergraduate students, doing research work on animal diseases and formulating methods for diagnosis and control, when adequate knowledge is acquired, it has the very important duty of aiding practitioners to an understanding of new methods of diagnosis, which have been developed to the point where they have become a part of professional equipment. It is reasonable for the State to conduct researches on which are based efficient methods for diagnosis and then turn these methods over to practitioners to apply. It does not seem wise for the State to take over for individuals the work that belongs to practitioners. On the other hand, if the new knowledge is not applied properly by local veterinarians or specialists in the field, animal owners will seek, and justly so, some means by which they may be benefited by the results obtained. The question before us is: "Who will make the tests?"

I hope there are men enough in the veterinary profession of New York State who will interest themselves in this work, to make it

possible for every flock and herd owner, who desires it, to obtain, with its correct interpretation, the agglutination test for the diseases in question. Competent local practitioners can relate the findings of the tests to conditions which are peculiar to the individual flock or herd, and can furnish the owner guidance based on this relation better than a stranger. Logically they are the best messengers to bring the glad news of better methods for disease control from the research laboratories and apply them in the flocks and herds of their clients.

INDEX

A

Abortion, human, 173–178; *see also Bang abortion disease*
Agglutination test, 103–105, 121–124, 178–181, 188–193; *see also bacillary white diarrhea, Bang abortion disease, Brucella abortus*
Ambulatory clinic, 12, 13, 27–30
Amidostomum anseris, 51
Anthrax, 7, 38
Appropriations, 19, 22
Autogenous bacterins, 39, 44
  vaccines, 44, 57

B

Bacillary white diarrhea, 50, 55, 56, 131–145, 188–193
Bacillus bovisepticus, 67–69
  pyocyaneus, 65
Bacterins, autogenous, 39
  Bang, abortion disease, 89, 90
Bacterium abortum, 81–102, 118–120
  anatum, 58
  gallinarium, 146
  pullorum, 50, 51, 131–172, 188, 189
  sanguinarium, 146–172
Bang abortion disease, 81–102, 188–193
  organism, *see Bacterium abortum*
Biliography, abortion, 177, 181
  agglutination test, 181
  bacillary white diarrhea, 143
  Bacterium bovisepticum, 172
  Bacterium pullorum, 145, 172
  blood, 80
  Brucella abortus, 130, 177, 181, 185
  Brucella melitensis, 177, 181, 185
  castration, 80
  goats, 80, 177
  hog cholera, 117
  infectious abortion, 130
  Malta fever, 177, 181
  undulant fever, 177
Blackleg, 7, 38
Blood, goats, 76–80
Brucella abortus, 36–38, 40, 41, 125–130, 173–185, 190
  melitensis, 173–177, 180, 182

C

Castration, 76–80
Cat, diseases, 33–35
Cattle, diseases, 27–32; *see also agglutination test, Bacterium abortum, Brucella abortus, infectious abortion, physical diagnosis, scabies*
Chicken pox, 56, 57
Clinical diagnosis, 61–69
Clinics, 12, 13, 18, 25–35
Complement fixation test, 189
Consulting clinic, 31, 32
Contagious abortion, 7; *see also infectious abortion*
Convulsions, 70–75
Cornell Veterinarian, 13, 16

D

Diagnosis laboratory, 18
Diarrhea, fowl, 50, 55, 56
Distemper, 75
Dog, diseases, 33–35, 70–75
Dourine, 7
Ducksm, *see poultry diseases*

E

Estrus equi, 65

F

Faculty, 8–10
Fellowships, 8, 9
Finances, 21, 22
Fowl diseases, 188–191; *see also bacillary white diarrhea*
  plague, 50
  typhoid, 58
Frights disease, 73

G

Geese, diseases, 51
Glanders, 7, 37
Goats, blood, 76–80

H

Haemonchus contortus, 76
Hemorrhagic septicemia, 7, 38
Hog cholera, 7, 106–117
Hookworms, 73, 74
Horse, diseases, 27–32
Horseshoeing, 14

I

Indigestion, 73, 74
Infectious abortion, 37, 38, 125–130; *see also Bang abortion disease*
  bronchitis, 57, 58
Influenza, 7
Instruction, 12–14
International Education Board, 8, 9

J

Johne's disease, 38

L

Laboratory diagnosis, 16, 17, 30, 36–49
Lecturers, 10, 11, 15, 16, 19, 20
Library, 15

M

Mallein, 44
Malta fever, 173–177, 180
Man, *see abortion, Bang abortion disease, Brucella abortus, undulant fever*

N

Needs of college, 19

O

Otorrhea, 72, 74

P

Parasites, 50, 51, 56; *see also convulsions*
Physical diagnosis, 61–69
Pigs, *see hog cholera*
Plant poisoning, 7
Plumbism, 51
Poisoning, *see plumbism, plant poisoning*
Poultry, 14
  diseases, 50–60; *see also bacillary white diarrhea, Bacterium pullorum, chicken pox*
Practitioners, 7
Publications, 13–16, 19, 20

R

Rabies, 37, 45
Research, 17, 18
Rickets, 71
Roundworm, 73, 74
Roup, 56, 57

S

Salmonella pullora, 55
Scabies, 7
Science and practice, 186–193
Serum, *see hog cholera*
Sheep, diseases, 27–32; *see also scabies*
Small animal clinic, 12, 13, 33–55
Southern cattle tick, 7
Students, 11, 12
Surgical clinic, 12, 31, 32
Swine diseases, 27–32; *see also hog cholera*

T

Tapeworm, 73, 74
Tuberculin, 39, 44, 45
Tuberculosis, 7
  avian, 50
Typhoid, *see Brucella abortus*

U

Undulant fever, 173–177

V

Vaccination, *see hog cholera*
Vaccines, Bang abortion disease, 89, 90
Veterinary Conference, 15, 16

THE LIBRARY OF THE
MAY 25 1927
UNIVERSITY OF ILLINOIS

UNIVERSITY OF ILLINOIS-URBANA
3 0112 119663760